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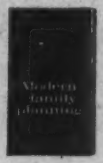

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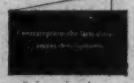

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY JULY 15 1961

PREMALIGNANT CONDITIONS OF THE VULVA, WITH PARTICULAR REFERENCE TO CHRONIC EPITHELIAL DYSTROPHIES*

BY

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[WITH SPECIAL PLATE]

Many authorities object to any condition being called premalignant, holding that cancer is a primary pathological change. Though what follows may be taken to lend a measure of support to this view, the title of this communication is a convenient cover for a consideration of those lesions, factors, and circumstances which are alleged to determine or favour the development of cancer of the vulva. The discussion is, moreover, limited to squamous-cell carcinoma, without regard to the rarer forms of malignancy such as sarcoma, melanoma, and adenocarcinoma, which together comprise less than 10% of all malignant growths of the vulva.

Factors in the Aetiology of Squamous-cell Cancer of the Vulva

Local Irritation

The development of cancer at any site can be determined by certain types of chronic tissue injury or "irritation." In the case of epithelioma of the vulva the only reasonably well established example of this is contact with carcinogenic mineral oils. Thus in 1932 Gerrard showed that women operatives in the cotton and woollen mills of Lancashire and Yorkshire, directly exposed to oil-impregnated waste materials of their trade, were twice as liable to suffer from cancer of the vulva as were women of similar ages engaged in other occupations. Repeated exposure to *x* rays and radium is an acceptable theoretical cause of malignant disease of the vulva, but has not, in practice, been shown to operate. It is, nevertheless, one of several good reasons for abandoning the use of radiation therapy for intractable pruritus vulvae. Direct trauma, at one time suggested by Taussig (1917, 1940), is, however, not acceptable because cancer of the vulva occurs as frequently in virgins and nulliparous women as it does in those exposed to coitus and childbirth.

Infections, Condylomata, and Granulomata

The fact that vulval cancer commonly begins in the region of the clitoris is sometimes attributed to irritation from collections of smegma at that site, but this is no more than conjecture. The same is true of irritation caused by chronic purulent discharges. Such discharges sometimes cause condylomata acuminata, which in the past were regarded as precancerous (Taussig, 1931, 1940). Though Stening and Elliott (1959) reported that cancer in four of their cases was superimposed on "papillomatous warts," most modern findings are opposed to the concept that condylomata can become malignant (McAdams and Kistner, 1958). Older reports to the contrary were probably based either on confusion between the histological appearances of condylomata and of cancer, or on the assumption that a papillomatous pattern of squamous carcinoma implies that an innocent papilloma has become malignant. Similarly, there is no convincing evidence to support an older impression that syphilitic and tuberculous lesions of the vulva can determine the development of cancer (Lunin, 1949; Stening and Elliott, 1959; McAdams and Kistner, 1958). Chronic inflammatory disease, especially if accompanied by hypertrophic tissue reactions, can simulate cancer both macroscopically and microscopically; this fact also makes it difficult to decide whether granuloma inguinale and lymphogranuloma can lead to epithelioma of the vulva.

Lunin (1949) reported that 60% of negresses suffering from vulval carcinoma had previously had granulomatous venereal disease, and Stewart (1959), besides finding support in the literature for the idea that granulomata can become malignant, reported six cases in which epithelioma of the vulva occurred in women suffering from either ulcerative or proliferative granulomatous lesions. Nevertheless McAdams and Kistner (1958), in their prospective study of seven women with vulval granuloma, did not encounter one case of cancer, so the matter remains unsettled.

Age

Squamous-cell cancer of the vulva, like skin cancer at any site, is a disease of old age. Involutionary changes associated with ageing in some way encourage mutations which can lead to malignant behaviour of the epithelial cells.

*Based on the Emily Blake Memorial Lecture delivered at Newcastle upon Tyne on April 12, 1961, under the auspices of the North of England Council of the British Empire Cancer Campaign.

Inherent Predisposition to Cancer

Differences in racial proclivities to cancer of the vulva are disputed (Rubin and Novak, 1956 ; Corscaden, 1956 ; Taussig, 1931), and, even if they could be demonstrated, would be open to several interpretations, because environmental as well as genetic factors might be concerned. Nevertheless, several writers point out that women with cancer of the vulva often have malignant disease at other sites, which appears either before or after the vulval condition and is unrelated to it. Thus Taussig (1940) noted that 10 out of 155 of his patients (6.4%) had malignant disease elsewhere. In the series reported by Green *et al.* (1958) coincidental malignant disease was present in 13% and there was a family history of cancer in 21%. Among the small series of 25 women suffering from cancer of the vulva treated by one of us (T. N. A. J.), four had malignant disease elsewhere. All such observations, however, are uncontrolled, and it might be possible to show that women of similar age who do not suffer from cancer of the vulva give similar histories.

Vulval Cancer as Part of a "Field Change"

Even though vulval cancer has not yet been shown to have a significant relationship to malignant disease elsewhere in the body, there is much to suggest that it is often merely one manifestation of a widespread field change in the epithelium of the vulva and even of the vagina. This explains why cancer of the vulva not infrequently presents as multiple separate primary lesions, a phenomenon noted by Berkeley and Bonney (1910) and confirmed by many subsequent observers. The not uncommon coexistence of multiple epithelial neoplastic changes is illustrated by the following two cases.

Case 1.—A. R., aged 67 years, complained of postmenopausal bleeding for two months and soreness of the vulva for four years. The skin around the clitoris and on one labium minus showed hyperkeratosis, but quite separate white patches were also seen on several areas of the vagina and on the cervix. Histological examination showed that all these lesions, from the cervix to the vulva, represented separate areas of intra-epidermal cancer with normal epithelium intervening. At one place in the vagina the cancer showed early invasion. Fifteen months after treatment by total hysterectomy, colpectomy, and simple vulvectomy, this patient presented with another lesion on the vulva which proved to be an early invasive cancer. Radical vulvectomy was then performed. Nine months later she was well and the skin covering the area where the vulva had been looked normal. Nevertheless, skin scrapings showed "cells with large nuclei which suggest dyskaryosis." She died three years after the original treatment from intrapelvic recurrence of the vaginal growth. At that time the "vulval" skin still looked macroscopically healthy.

Case 2.—R. L., aged 58 years, complained of an ulcer on the left labium minus which had been present for a few months. Ten years previously she had been treated successfully for carcinoma of the cervix by radiotherapy. Seven years after this she developed an epithelioma of the anus, which was treated by wide excision and block dissection of the inguinal nodes. The vulval lesion, which appeared three years later, was a new and separate intra-epidermal cancer with possible early invasion of the underlying stroma at one point. This patient died during the induction of anaesthesia before any surgery was undertaken. Necropsy showed no evidence of cancer in the cervix, vagina, or anal and rectal canals. Only the vulval tumour was present.

A diffuse epithelial instability not only accounts for a multifocal origin of cancer of the vulva, but may well explain certain "recurrences" of cancer after radical vulvectomy. It is all too often assumed that the "recurrence" represents an incomplete excision of the original growth. Sometimes, however, it is truly a new growth, unrelated to the original growth except in so far as it is another manifestation of an underlying field change. This was recognized by Taussig (1940), four of whose patients developed fresh tumours of the vulva several years after radical vulvectomy for cancer. The following is another example.

Case 3.—A. T., aged 68 years, complained of itching and soreness of the vulva for four years and one year respectively. She had a papillomatous lesion on one labium majus with evidence of hyperkeratosis elsewhere. Radical vulvectomy was performed and cancer was found only in the papilloma ; the epithelium from all other areas was not malignant. Two years later another vulval cancer appeared and was excised widely. The tissues then showed widespread intra-epidermal cancer with invasion beginning at one point. Within another eight months the skin immediately anterior to the anus, previously healthy, showed cancer *in situ*.

Leucoplakia and Leucoplakic Vulvitis

A diffuse change in the vulval skin which is most often accepted as being premalignant, or at least as indicating that the woman has an increased risk of developing cancer, is leucoplakia. Unfortunately there is no agreement among either clinicians or pathologists as to what constitutes leucoplakia, and as to its relationship to alleged other diseases such as kraurosis, primary atrophy, leucoplakic vulvitis, and lichen sclerosus vel atrophicus. It is therefore necessary to examine the evidence which justifies the use of these terms to indicate separate disease entities before considering what truly sequential relationship they bear to frank carcinoma.

The word "leucoplakia" is derived from *leucos*—white, and *placos*—a flat plate. It was first used in medicine to denote a skin condition which is characterized by a naked-eye appearance of "white plaques," and it was applied originally to the tongue and mouth. Indeed, its early use was restricted to such an appearance occurring on mucous surfaces or mucocutaneous junctions (Hunt, 1954). Because of this restriction Berkeley and Bonney (1909), and later Hunt (1940, 1954), preferred the term "leucoplakic vulvitis" when the white plaques occurred outside a mucocutaneous junction. But neither "leucoplakia" nor "leucoplakic vulvitis" was ever originally intended to denote anything more than a clinical picture of white plaques, the plaques representing an increased deposition of keratin on the skin surface. Beneath this keratin various histological changes in the epidermis and dermis were and are described, with the possibility of at least two directly opposing pictures—namely, hypertrophy and atrophy (Special Plate, Figs. 1, 2, 5, 6). In the absence of any clear microscopical criteria the cautious pathologist has therefore long refused to accept leucoplakia as a histological diagnosis. In practice he is wise if he says no more than that the microscopical appearances of vulval skin submitted for examination are not incompatible with the clinician's diagnosis of leucoplakia.

The clinician, on the other hand, while once content to diagnose leucoplakia or leucoplakic vulvitis according to what he saw at the bedside, is, for reasons which

will become apparent, now reluctant to assume responsibility. The tendency therefore is for the gynaecologist to say that leucoplakia is a histological diagnosis and for the histologist to say that it is a clinical diagnosis. Historically and etymologically the latter is right. Nevertheless, some clinicians and pathologists (Wallace and Whimster, 1951; Wallace, 1955; McAdams and Kistner, 1958) now restrict the diagnosis of leucoplakia to those vulvas which, irrespective of their naked-eye appearances, show on section such a degree of disorderly epithelial activity ("atypism") as to make it approximate to malignant change (Special Plate, Figs. 3 and 4). Wallace and Whimster (1951) went so far as to refer to "the state of premalignant hyperplasia known as leucoplakia," at the same time admitting that they established their criteria for the histological diagnosis of leucoplakia by studying sections of 20 cases of squamous-cell carcinoma of the vulva and noting the appearances of the adjacent epithelium. This retrospective approach not only makes it easy to show that leucoplakia is related to cancer, but also makes it a comparatively rare histological disease in which the vulval skin does not always look white and thickened to the naked eye. Furthermore, it creates confusion between leucoplakia and intra-epidermal cancer, as was noted by McAdams and Kistner (1958).

This new concept of leucoplakia is not only out of keeping with the name and its origin; it is entirely different from that of Berkeley and Bonney (1909) and of Taussig (1931). While describing certain variable histological features such as thickening or thinning of the epithelium, lengthening or shortening of the rete pegs, and hyaline change beneath the epidermis, these authors, and their contemporaries, paid no particular attention to abnormal cellular activity as manifested by pleomorphism, hyperchromatic nuclei, frequent mitoses, and loss of stratification. Moreover, they regarded the macroscopic appearances as of first importance. Thus, Taussig (1931) said leucoplakia "is characterized by the formation of whitish plaques of thickened epidermis, with sclerosis of the subjacent connective tissue and absence of elastic fibres producing a parchment-like consistence of the affected skin." And it is leucoplakia as defined by these older writers which most gynaecologists believe to be premalignant—a belief which they consider justifies the almost routine performance of vulvectomy for any chronic skin lesion showing naked-eye signs of hyperkeratosis. Any doubts they may now have about these matters are due to the confusion which has arisen over other terms such as kraurosis, primary atrophy, and lichen sclerosus vel atrophicus. These are supposed to be separate clinical and pathological states despite the fact that they produce skin changes which can and do pass for leucoplakia. Nevertheless, views on these are equally variable and uncertain.

Kraurosis Vulvae

Disagreement about kraurosis extends even to the meaning of the word. It derives from *krauros*, meaning "brittle," yet Taussig (1922) and Novak and Novak (1952) said it means "shrinkage," while Hunt (1940, 1954) reached the conclusion that it means "white and dry." The last interpretation is incompatible with "kraurosis rouge," one of several forms of kraurosis described by Jayle (1906); the second explains the traditional British view that kraurosis is a condition affecting only the introitus, causing contracture and therefore dyspareunia rather than pruritus. Kraurosis, differs, it is said, from leucoplakia in that its epithelial changes are characterized by atrophy and in that cancer never supervenes. These ideas follow the teaching of Berkeley and Bonney (1909) and Bonney (1938), but have never been accepted outside Great Britain. The American concept was formulated by Taussig (1922, 1931) and envisages contracture of the introitus merely as a possible complication of leucoplakia, a result of excessive degenerative changes in the epidermis and dermis. This relating of kraurosis to the atrophic phase of leucoplakia is attractive if only because it divorces kraurosis from senile atrophy (which is physiological), and permits an explanation of those cases occurring in youth and while ovarian function is present. It also conforms to a large extent with ideas on kraurosis which have been held by workers on the Continent of Europe throughout this century (Jayle, 1906; Darier, 1928).

Taussig (1922, 1931) regarded kraurosis as complicating 10% to 20% of cases of leucoplakia, and in 1917 attributed as many vulval cancers to pre-existing kraurosis as to leucoplakia. Rubin and Novak (1956) reversed Taussig's thesis and said that kraurosis can progress to leucoplakia and hence to cancer. This fits in more with the concept of dermatologists such as Wallace and Whimster (1951) and Wallace (1955), who consider that kraurosis vulvae, or primary atrophy as they prefer to call it, changes to leucoplakia within ten years in 50% of cases, and that, with or without this intermediate change, the development of cancer is a real possibility. Nevertheless, a background of traditional British opinion is still evident in Wallace's (1955) definition of primary atrophy as "a sclerosing progressive atrophy of the mucocutaneous integument of the vulva, with a characteristic histology." When it comes to describing this characteristic histology, however, difficulty arises, for the appearances overlap with those of atrophic leucoplakia and of lichen sclerosus.

Lichen Sclerosus vel Atrophicus

Lichen sclerosus, as a cause of a whitish change in vulval skin and of pruritus, was introduced to the gynaecologists by the dermatologists comparatively recently, and some are in danger of being persuaded that, no matter what view may be taken in regard to kraurosis (primary atrophy), there is a clear clinical and pathological difference between leucoplakia and lichen sclerosus. What is the evidence for this? Hunt (1940, 1954) says that lichen sclerosus is a vulval form of lichen planus and that it is identical with leucoplakic vulvitis, or at least with the atrophic form of this disease. Wallace and Whimster (1951) and Wallace (1955), however, are equally certain that lichen sclerosus is "an atrophic form of scleroderma unrelated to lichen planus and occurring in various parts of the body, frequently in the perianal area and the vulva." From this sort of statement has evolved the idea that leucoplakia is a change limited to the vulva, and that when a white skin change is seen to affect the perianal areas the explanation is lichen sclerosus.

Apart from this alleged anatomical difference, it may be difficult, if not impossible, to distinguish lichen sclerosus from leucoplakia on naked-eye examination. Yet histological differences are claimed, the main one being that the epithelium is atrophic rather than hypertrophic. Otherwise the stated characteristics are

hyperkeratosis, disappearance of elastic tissue, hyalinization of the collagen layer, and subepithelial infiltration with leucocytes. What is the difference between this and the so-called atrophic form of leucoplakic vulvitis? We agree with Hunt; there is none. What are the differences between this and primary atrophy or kraurosis? The only ones claimed by Wallace (1955) are that in primary atrophy there is less deposition of keratin and less destruction of collagen, but these are mere quantitative distinctions, and they disappear in advanced cases (Special Plate, Figs. 2 and 5). Hunt (1940, 1954) considered that kraurosis is no more than a late stage of lichen sclerosus, and Stening and Elliott (1959) inclined to a similar view. Wallace (1955), on the other hand, concluded that lichen sclerosus can change to leucoplakia, and, in his experience, in 25% of cases the latter begins as lichen.

Those who consider leucoplakia strongly precancerous admit that malignant change is rarely seen in the perianal skin. This, it is argued, is because the perianal changes are due to lichen sclerosus. And there are some dermatologists who consider that one of the essential differences between lichen sclerosus and leucoplakia is that the former never becomes malignant. This, however, is not the view of Wallace and Whimster (1951) and Wallace (1955), who considered that lichen sclerosus carries a real risk of malignancy, possibly because of an intermediate leucoplakic change. Stening and Elliott (1959) also commented that a case diagnosed as lichen sclerosus sometimes shows "atypical" epithelial activity.

The above considerations can be summarized by saying that, according to various authorities:

Lichen sclerosus is or is not the same as leucoplakic vulvitis.

Lichen sclerosus is or is not related to lichen planus.

Lichen sclerosus is or is not related to scleroderma.

Lichen sclerosus can or cannot change to leucoplakia.

Lichen sclerosus is or is not premalignant.

Lichen sclerosus can or cannot change to kraurosis (primary atrophy).

Leucoplakia is or is not a clinical diagnosis.

Leucoplakia is or is not a histological diagnosis.

Leucoplakia presents two or more different histological pictures.

Leucoplakia is or is not a histological state of atypical epithelial hyperplasia bordering on malignancy.

Leucoplakia can or cannot supervene on lichen sclerosus.

Leucoplakia can or cannot supervene on kraurosis.

Kraurosis (or primary atrophy) is or is not a separate disease.

Kraurosis is or is not the end-result of leucoplakia or of an atrophic form of leucoplakia.

Kraurosis is or is not the end-result of lichen sclerosus.

Kraurosis does or does not have a characteristic histological appearance.

Kraurosis is or is not premalignant.

Thus critical appraisal of the literature as a whole and, indeed, of individual articles on these conditions leads to the conclusion that, unless it be accepted that leucoplakia is a purely histological finding of disorderly epithelial activity bordering on malignancy—a concept quite foreign to traditional ideas—there are no essential clinical or histological differences between kraurosis (primary atrophy), leucoplakia, and lichen sclerosus vel atrophicus. Any alleged clinical distinctions are no more than individual impressions and opinions; any supposed histological differences are no more than variations in epithelial responses to some unknown factor. Indeed, microscopical pictures which would be accepted individually as representing each of these conditions can often all be found in the same vulva. To give them each a special name does not make them into separate diseases. Indeed, there is a real danger that the effects of a disease are being mistaken for the disease itself, and ignorance is being concealed by superficially imposing labels with classical roots. This view is all the more justified by the fact that the various protagonists of leucoplakia, leucoplakic vulvitis, lichen sclerosus, primary atrophy, and kraurosis find it impossible to state the specific cause for any of what they regard as different diseases.

If progress is to be made in this admittedly difficult field the time has come for it to be freely recognized that the vulval and perianal regions are subject to chronic skin changes, the particular characteristics of which are probably conditioned by environment rather than by causes. The fact that the vulva reacts to various toxic and adverse agents differently from the skin elsewhere is almost certainly explained in part, if not in whole, by its circumstances. Thus, its peculiar tendency to show hyperkeratosis is probably due to heat, moisture, and lack of aeration. A similar reaction is seen in the skin affections occurring between the toes, sites which offer the same environmental conditions.

The evidence suggests that not only can the same aetiological factor cause different types of epithelial reaction in the vulva, but that different aetiological factors can be the foundation of similar vulval changes (Jeffcoate, 1955, 1957) (Special Plate, Figs. 1, 2, 5, 6). It therefore seems more important to discover the underlying causes for the epithelial changes than it is to produce labels which purport to describe the different macroscopic or microscopic features of these skin reactions, and to denote separate diseases. Meanwhile, and irrespective of their appearances, all these intractable skin changes for which a specific cause is not clear are best given clinically an all-embracing and non-committal title such as "chronic epithelial dystrophy." The pathologist equally should be untrammelled by the need to append precise dermatological nomenclature to his report. His factual description of the various histological findings should, nevertheless, include an assessment of the activity and orderliness of the squamous epithelium, for it is these which will guide treatment.

Risk of Cancer Supervening on Chronic Epithelial Dystrophy

The traditional view of the seriousness of macroscopically diagnosed leucoplakia (or leucoplakic vulvitis) as a forerunner to cancer stems mainly, in Great Britain from the writings of Berkeley and Bonney (1909) and Bonney (1908, 1938), and in the New World from those of Taussig (1917, 1922, 1930, 1931, 1940). The former writers did not attempt to assess the statistical chances of such a development, whereas Taussig (1930) concluded that 50% of women diagnosed clinically as suffering from leucoplakia are destined to develop cancer within the subsequent ten years. Indeed, at other times (Taussig, 1922, 1931) he went so far as to say

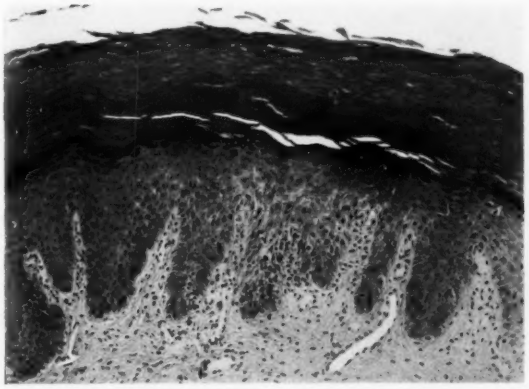

FIG. 1.—"Hypertrophic leucoplakia," showing hyperkeratosis and acanthosis of the epithelium. The dermis has a hyaline appearance. (×75.) This section is from the vulva of a woman suffering from chronic moniliasis and previously treated unsuccessfully by simple vulvectomy.

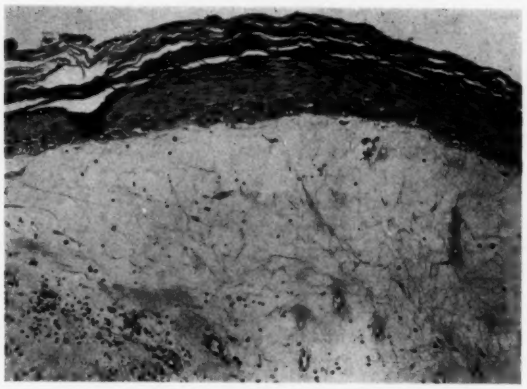

FIG. 2.—"Atrophic leucoplakia." A moderate degree of hyperkeratosis is associated with a thin epithelium devoid of rete pegs. A subjacent hyaline zone in the dermis is conspicuous. (×75.) The patient in this case suffered from fungus infection of the feet and was considered to have a trichophytid reaction on the vulva.

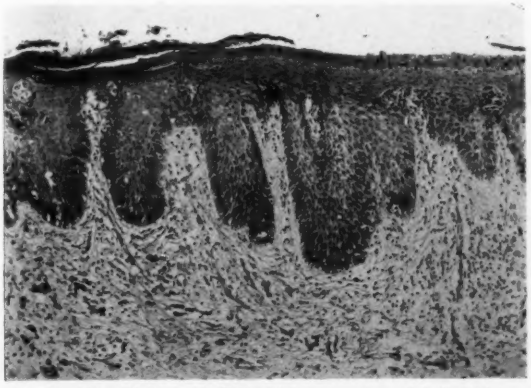

FIG. 3.—Disorderly epithelial activity sometimes labelled histological leucoplakia. The keratin layer is thin, but there is considerable irregularity and prolongation of the rete pegs into the dermis. (×75.)

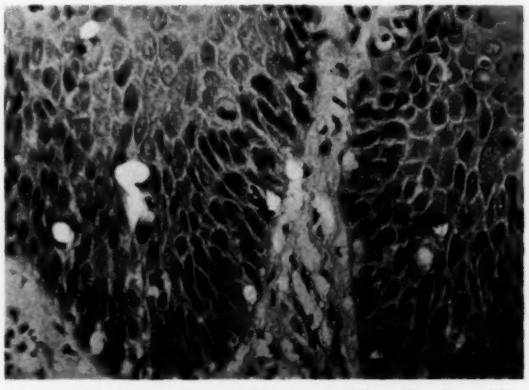

FIG. 4.—High-power view of Fig. 3. (×288.) (See Case 6 in text.) This section was initially reported as showing a benign leucoplakic appearance, but the epithelium shows irregular cell activity and frequent mitoses, a picture approaching cancer *in situ*. The patient developed invasive cancer 10 years after excision of the lesion illustrated here.

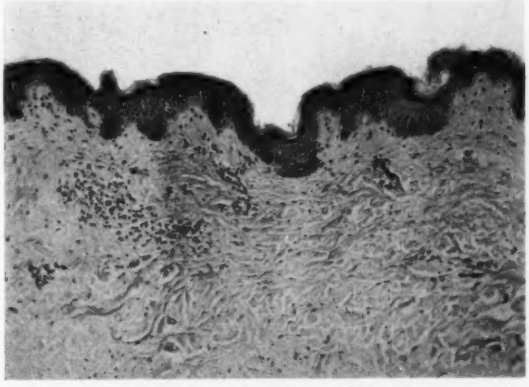

FIG. 5.—Lichen sclerosus vel atrophicus. Slight keratinization is associated with a thin and inactive epithelium. Though diagnosed histologically as lichen sclerosus, the appearances correspond equally well to descriptions of atrophic leucoplakia. (×70.) This specimen came from a patient whose vulval dystrophy was subsequently proved to be caused by diabetes mellitus.

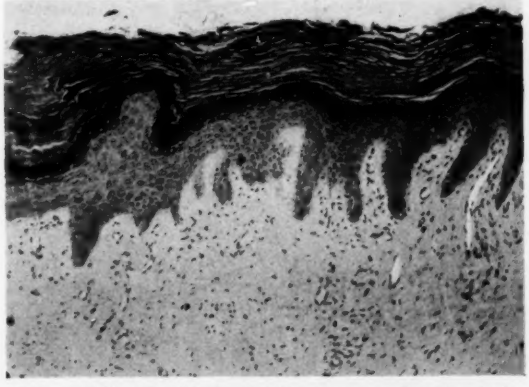

FIG. 6.—"Hypertrophic leucoplakia," showing hyperkeratosis, long rete pegs, and hyalinization of the subepithelial zone. (×70.) This vulval skin was obtained from a patient suffering from neurodermatitis intermittently for 20 years. There were lesions on the arms, legs, and neck as well as the vulva.

D. ABRAHAMS AND W. BRIGDEN: MITRAL INCOMPETENCE AND PULMONARY HYPERTENSION

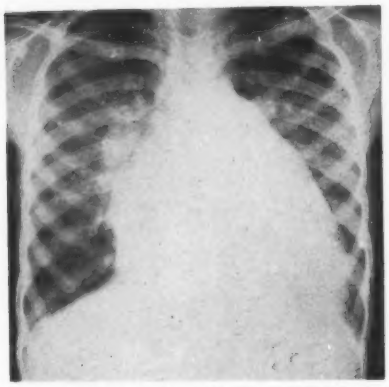

FIG. 1.—Chest radiograph of a girl aged 16 with mitral incompetence and pulmonary hypertension. The aorta is inconspicuous; main pulmonary arteries and outflow tract of right ventricle enlarged.

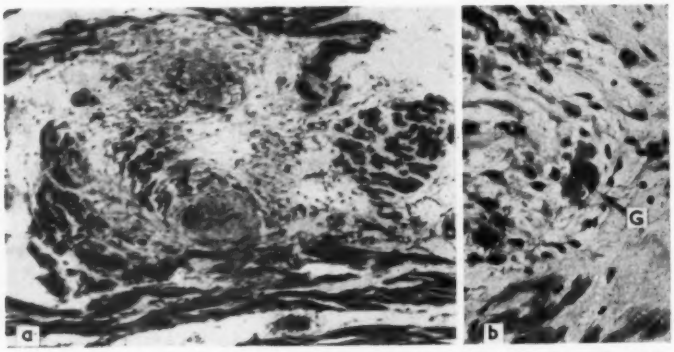

FIG. 3.—(*a*) Aschoff nodule showing collagen debris. (×76.) (*b*) Aschoff nodule showing one of the rare giant cells (G). (×236.)

FIG. 2.—Radiograph showing a late stage of mitral incompetence and pulmonary hypertension when tricuspid incompetence has supervened. Right auricle is large; peripheral vascular markings are diminished.

FIG. 4.—Aschoff nodules adjacent to a small vessel. (×228.)

L. S. TAITZ *ET AL.*: ACUTE NEPHRITIS ASSOCIATED WITH CONGENITAL SYPHILIS

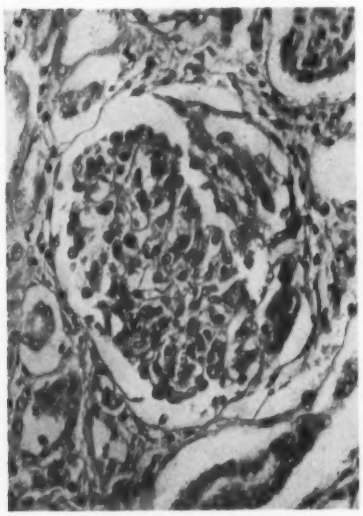

FIG. 1.—Glomerulus from kidney of Case 1, showing hypercellularity and adhesion between tuft and Bowman's capsule. (H. and E. ×260.)

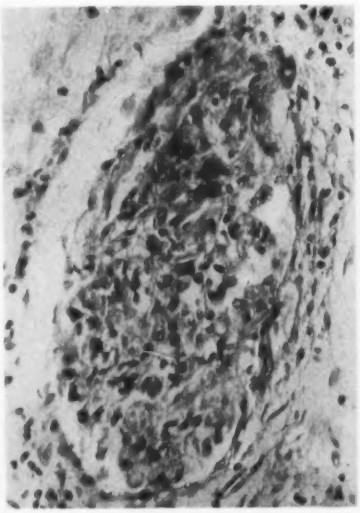

FIG. 2.—Markedly swollen glomerulus. (H. and E. ×260.)

FIG. 3.—Eosinophilic necrosis of glomerular tuft. (H. and E. ×260.)

that leucoplakia inevitably turns to epithelioma provided the sufferer lives long enough. Conversely, and on the same evidence, he stated that at least one-half of all cases of squamous-cell cancer are preceded by leucoplakia. But these views, so widely accepted, were based on indirect or prejudiced evidence. The main supporting argument was that in cases of established cancer it was possible to demonstrate in adjacent tissues histological signs of leucoplakia. This is not surprising, since at the time in question Taussig recognized two quite opposite histological pictures in leucoplakia, one showing underactive and the other overactive epithelium, while Berkeley and Bonney described no fewer than four different microscopical appearances. More recent writers, such as Way (1951), Langley *et al.* (1951), Corscaden (1956), Bibby (1957), Green *et al.* (1958), Stening and Elliott (1959), and Rewell (1960), also base their assessments of the frequency of coincidental leucoplakic changes in the vulva of women suffering from cancer on differing and often multiple histological criteria, so the reported incidence of the association varies from 12% to 80% of cases. The estimate that cancer develops in 50% of cases of leucoplakia was reached on the basis that, of specimens of vulval skin submitted to histological examination, there are equal numbers of cancers and leucoplakias (Taussig, 1930; Langley *et al.*, 1951; Novak and Novak, 1952). This takes no cognizance of the many cases of leucoplakia not subjected to biopsy.

When leucoplakia was found in association with established cancer it was, and is, assumed that it preceded the growth, and to bolster this idea it is pointed out that women with cancer often give a long history of pruritus vulvae. Yet even Taussig (1940) reported that only 42 out of 155 women (approximately 30%) with vulval carcinoma ever complained of pruritus, and that of unspecified duration. Indeed, all observers except Stening and Elliott (1959) record that pruritus is not present in more than 40% of cases, and this is confirmed by our experience. Only eight out of a consecutive series of 25 women treated for vulval carcinoma by one of us (T. N. A. J.) had ever had pruritus, the duration being less than one year in two cases.

The final argument in support of the idea that leucoplakia antedates malignant disease is based on age incidences, the difference at the time of treatment averaging 10 to 15 years (Taussig, 1930; Langley *et al.*, 1951).

Despite these indirect and retrospective evidences that clinical leucoplakia is a premalignant state, most gynaecologists admit that they have rarely, if ever, seen malignant change occur in a patient under observation. Even Taussig (1940) reported that during his lifetime he had seen only three women develop cancer of the vulva while under medical care. In two of these the original lesion had been studied histologically, but in only one is it mentioned that the microscopical appearances were those of "leucoplakia." In the third case biopsy was not undertaken, so the condition could well have been cancerous from the onset.

Unfortunately, few prospective or follow-up investigations are reported. Hunt (1940), however, said she had studied 96 cases of lichen sclerosus (the same as leucoplakic vulvitis to her) over a prolonged but unspecified period, and cancer developed in only four. She did not mention whether the presence of cancer was excluded by biopsy when these patients were first seen, and this is important. Langley *et al.* (1951) studied women who had been treated by simple or partial vulvectomy for "leucoplakia," and in all cases histological examination was carried out. Of these cases 122 were followed for more than five years, and 72 (59%) had a recurrence of leucoplakia or pruritus, or both. Yet, of them all, only one developed cancer of the vulva, and that 12 years after the original vulvectomy. By that time the patient had had four operations for recurrent leucoplakia. Böttger and Dittmann (1957) followed 82 women suffering from "chronic pruritus" and 78 suffering from "kraurosis" (Continental definition) for varying periods up to eight years. Among the chronic pruritus group there were no cases of cancer, but in the kraurosis group seven women developed malignant disease. These writers pointed out, however, that a 9% incidence of skin cancer is the same as might be expected for any chronic dermatosis occurring in any part of the body amongst old women. A similar conclusion was reached by McAdams and Kistner (1958) in their valuable study of 397 women subjected to simple vulvectomy or biopsy for various diseases of the vulva. Altogether 201 women with chronic dermatoses (with or without sclerosus) were followed, 136 of them for more than three years. None developed cancer, nor did any of those who were classified separately as suffering from lichen sclerosus, granuloma, and condylomata. Cancer occurred only among the 32 women classified as suffering from leucoplakia, the criterion for this diagnosis being a histological finding of disorderly overactivity of the epithelium. These women were in fact suffering at the outset from skin changes to be regarded as near-malignant, eight of them from border-line cancer. Twenty-seven were followed for periods varying from three to 25 years, and only three developed cancer. Among this particular group the incidence of cancer was therefore 10%, the same figure as for any senile dermatosis in any site.

Our experience is similar. During 25 years one of us (T. N. A. J.) investigated and treated 117 women suffering from chronic epithelial dystrophy of obscure aetiology. This represents less than 5% of patients complaining of pruritus, for all cases in which an obvious cause such as trichomoniasis, moniliasis, and known diabetes was present are excluded. To be included pruritus had to be of long duration, intractable to simple remedies, and associated with obvious vulval skin changes. An attempt was made to follow up all these women, but this failed in 14 cases, leaving 103 in which the after-history is known. Ten of these women had died, but the condition of the vulva at the time of death is recorded. The periods of time during which these women were observed are as follows:

Less than 1 year	7 cases	22
1–3 years	15 ,,	
3–5 ,,	15 ,,	81
5–15 ,,	54 ,,	
15–25 ,,	12 ,,	
	Total cases	103

Twenty of the patients had a vulval lesion which was essentially red or purple in colour, with or without ulceration; in the remainder the skin showed hyperkeratotic white patches of varying size and distribution with or without intervening red areas. All were investigated, and the over-all conclusions as to the cause of the dystrophies are to be described in detail elsewhere.

Histological examination of the skin was carried out in 65 cases, and revealed intra-epidermal cancer in four and early invasive cancer in one. Abnormal and disturbing epithelial basal-cell activity was present in another two. The five cases in which cancer was clearly present at the outset are set aside, and the findings in regard to the subsequent development of malignant disease relate to the remaining 98 women. Twenty-seven of these were treated by simple or partial vulvectomy (three of the operations being performed in clinics other than our own), and 13 had a recurrence of the vulval dystrophy while under observation. The remaining 71 patients, including nine who had already had an unsuccessful vulvectomy operation before consulting us, were treated conservatively.

Only three of the 98 women (3%) have so far developed cancer, and the details of their cases are as follows:

Case 4.—P. S., aged 54 years, complained of soreness of the vulva for one week. The perianal skin was white and thickened, the vulva looked atrophic with red areas of excoriation. Biopsy was not carried out; local applications were prescribed empirically and all symptoms subsided quickly. She reported more than two years later, having had a return of soreness during the previous eight months. The whole vulva was then red and there was a malignant ulcer on one labium majus. Radical vulvectomy was performed, but the patient died from spinal metastases one year later.

Case 5.—A. D., aged 39 years, had had pruritus vulvae for several years. The skin of the vulva, perineum, and perianal regions was the colour of raw beef with areas of superficial ulceration. Biopsy was not performed, and the skin reaction was thought to be an allergic one. Symptoms appeared to improve with medical treatment, and the patient defaulted from the clinic, only to return seven months later with a malignant ulcer on one labium majus. By that time the red reaction had disappeared; hyperkeratotic white areas were present on the labia majora, labia minora, and around the clitoris. Radical vulvectomy was carried out, and the patient is alive and well seven years later.

This case is of importance in that it illustrates that leucoplakic appearances which are commonly seen alongside cancer may well represent a simultaneous and not a preceding change. When first seen this patient showed no sign of hyperkeratosis of the vulva; this only appeared later, at the same time as the overt cancer.

Case 6.—R. O., aged 35 years, complained of pruritus vulvae for 1½ years, and was found to have two localized thick white patches of skin, one around the clitoris and the other on the fourchette. Both were excised, and sections showed what was then described as "leucoplakia." Ten years later the patient reported to another hospital, saying that she had had discharging warts for four years. This condition proved to be a papillomatous squamous-cell cancer arising in the region of the clitoris. Radical vulvectomy was carried out, and the patient is alive and well four years afterwards—that is, 14 years after the original partial vulvectomy.

This history suggests low-grade malignancy, but what is more interesting is that review of the sections prepared from the skin originally excised revealed disorderly overactivity of the epithelium of a degree approaching that of carcinoma *in situ* (Special Plate, Figs. 3 and 4).

Thus, of the three women who developed invasive cancer, two did not have an original biopsy, and one when first seen had such epithelial activity as, in retrospect, to warrant a diagnosis of borderline malignancy. The occurrence of cancer in the last is easily understood; the other patients could well have had similar warning epithelial changes when first seen. They may even have had intra-epidermal carcinoma, which often gives the vulva a red appearance. For example, two of the four patients excluded from follow-up because they had cancer *in situ* had initially the typical red lesions of "Bowen's disease" (Jeffcoate *et al.*, 1944).

The conclusion is that none of our patients who we are certain had no histological changes suggestive of malignancy at the outset developed carcinoma of the vulva during many years of observation. This tallies with the findings of McAdams and Kistner (1958), who also make the point that atypical cellular activity which some workers are now labelling "leucoplakia" may already be cancer *in situ*. The subsequent development of invasive cancer is therefore not necessarily ever a complication of chronic epithelial dystrophy. Indeed, it would appear that many, if not all, of those women who develop cancer of the vulva after a long history of vulval dystrophy have had cancer or something approaching cancer from the onset of symptoms. Those whose dystrophy shows other histological features do not have a special tendency to develop cancer.

This means that clinical leucoplakia is not a premalignant state, and that the only vulval dystrophy which is is one which, on histological examination, shows disorderly epithelial activity. This can lie beneath a hyperkeratotic surface or beneath one which is practically devoid of keratin (Special Plate, Fig. 3). Indeed, it is probably more likely when the skin is not becoming keratinized, since this property is, in general, a sign of good cellular differentiation. Naked-eye examination therefore gives no indication whether a particular dystrophy carries a threat of cancer.

Management of Cases of Epithelial Dystrophy

It follows from the above that two procedures are essential to the management of the woman suffering from a chronic epithelial dystrophy of the vulva. The first is to search diligently for an underlying cause such as dietetic errors; achlorhydria; deficiencies of iron, riboflavin, folic acid, and vitamin B_{12}; allergy to clothing, cosmetics, toilet preparations, and other items; skin diseases not peculiar to the vulva; hyperglycaemia; glycosuria; and particularly fungus infections of the hands and feet, which can probably cause trichophytid lesions of the vulva and perianal skin (Jeffcoate, 1949, 1955, 1957) (Special Plate, Figs. 1, 2, 5, 6). The second is to carry out biopsy of the vulval skin, and this irrespective of what aetiological factor may be found on investigation. Even diabetes can cause an epithelial dystrophy (additional to diabetic vulvitis) which can be followed by cancer (Green *et al.*, 1958; Stening and Elliott, 1959). Omission to carry out biopsy in all cases almost certainly accounts for the fact that we were unaware that two of our patients who subsequently developed cancer were at risk.

McAdams and Kistner (1958) and Stening and Elliott (1959) emphasized that any senile keratotic lesion of the vulva warrants biopsy, but we would go further and say the same for any chronic dystrophy, irrespective of its naked-eye appearance and no matter whether it looks red, purple, white, grey, or yellow. Moreover, biopsy should be carried out even if the patient is premenopausal.

There are, however, two problems associated with biopsy and its interpretation.

(1) Carcinoma of the vulva is said to arise just as frequently in atrophic as in hypertrophic epithelium (Langley *et al.*, 1951; Wallace, 1955). Taussig (1930) said that 40% of vulval cancers begin in atrophic leucoplakic vulvitis. Such statements, however, are now subject to real doubt. The follow-up results of Langley *et al.* (1951) gave no support to the idea, while McAdams and Kistner (1958) clearly showed that cancer did not develop in any vulva which had not originally showed atypical epithelial overactivity. Our experience tallies.

(2) The epithelial appearance may vary from one part of the vulva to another, and the biopsy specimen does not necessarily reflect what may be happening elsewhere. The presence of hyperkeratosis, or of any other special naked-eye appearance, is an unreliable guide to the surgeon in determining which area of the dystrophic vulva is most likely to show abnormal cellular activity. Widely spaced multiple biopsies are therefore always necessary, and, even if the findings are negative, safety is added by keeping the patient under observation at three-monthly intervals, with possibly repeat biopsy if the dystrophy persists.

In an attempt to preclude the possibility of missing the significant field during biopsy, we tentatively examined vulval scrapings obtained by a technique similar to that of Ayre's for the cervix. This method, however, is unsatisfactory, because it is unusual for the smears to contain anything more than surface keratin or keratinized squames; the underlying and more important basal epithelial cells do not appear. It may be argued that the difficulty is avoided by carrying out simple vulvectomy in all cases of chronic epithelial dystrophy. If the skin change is strictly localized there is certainly something to be said for its complete excision as the best method of biopsy. But comparatively few dystrophies show enough localization to permit this; even vulvectomy is not enough to eradicate a skin lesion which is spreading to the groins, to the perineum, and around and behind the anus. Moreover, the pathologist, when presented with the whole vulva, has the same difficulty as the clinician in deciding which areas to choose for histological examination.

Unless the lesion is strictly circumscribed, partial or simple vulvectomy has little to commend it. According to the findings of McAdams and Kistner (1958), as well as those reported here, intra-epidermal or early invasive cancer will be found in 2% to 5% of cases of chronic epithelial dystrophy. For these the operation may be inadequate. Of the remainder, only 4% to 8% will show histological evidence of such epithelial unrest as to threaten cancer, the threat being real in probably not more than one out of ten of this small special group. Vulvectomy is therefore only justified on the grounds of preventing cancer in a very small proportion of cases, and these can be chosen by preliminary biopsy. Nevertheless, even without a cancer threat, it is often said that vulvectomy is justified because its results are good and because no other treatment is effective (Rubin and Novak, 1956; Corscaden, 1956). The latter statement is not correct according to our experience (which is to be reported elsewhere), and the former is no more than a general impression unsupported by published post-operative studies.

Apart from the fact that vulvectomy is mutilating and is frequently followed by dyspareunia, it does not cure the dystrophy or its symptom of pruritus in approximately 50% of cases. Thus, in the series reported by Langley *et al.* (1951) the recurrence rate among patients who had "leucoplakia" treated by partial or total vulvectomy was 59%. The figures of McAdams and Kistner (1958) are in keeping. For those women observed for three or more years after surgery the recurrence rates were:

For chronic dermatoses	52 out of 118 (44%)
,, lichen sclerosus	7 ,, ,, 11 (63%)
,, leucoplakia (atypical epithelium—precancerous)	7 ,, ,, 17 (41%)
Total ..	66 ,, ,, 146 (45%)

It is sometimes said that such high recurrence rates mean inadequate surgery. In fact, as mentioned above, unless the surgeon is prepared to excise skin out to the groins and from around the anus, it is often impossible to remove all the affected area. In our series excision was avoided unless the lesion was so limited and demarcated that it could be removed with ease. Even so our results are not good. Nine of the 103 women whose study is complete had previously had vulvectomy carried out unsuccessfully in other clinics. Among the remainder, simple vulvectomy was carried out 15 times and partial vulvectomy 12 times. Of these 27 women treated surgically, 13 suffered a recurrence of the dystrophy and one developed cancer.

Summary and Conclusions

Except for the repeated application of carcinogens such as mineral oils, and possibly for chronic granulomatous venereal disease, a local cause for squamous-cell cancer of the vulva is not known. Like skin cancer in general, involutionary changes associated with ageing are probably important predisposing factors.

Invasive cancer often begins by way of a field change which may be widespread and in which disorderly epithelial activity is the characteristic histological change. This activity is sometimes so pronounced as to justify the term "cancer *in situ*," but in lesser degrees should not be labelled "leucoplakia," for it bears no constant relationship to hyperkeratinization, which is implicit in this name.

There is as yet no evidence to justify the view that leucoplakia, leucoplakic vulvitis, kraurosis (primary atrophy), and lichen sclerosus vel atrophicus are separate disease entities. Judged by either macroscopical or microscopical criteria, these represent no more than a variety of skin reactions to adverse factors which, although obscure, are more likely to be "general" than "local." The type of reaction is dependent on the environment of the vulva and not on the underlying cause. It is therefore suggested that all these names should be abandoned, since they hinder rather than help the elucidation of their causes. In their place, and pending further knowledge, the non-committal inclusive term of chronic epithelial dystrophy is proposed as one convenient for clinical practice.

Clinically, carcinoma *in situ* can simulate a chronic epithelial dystrophy, and can be excluded only by biopsy. Histological examination of all dystrophies reveals abnormal and disorderly epithelial activity in only 4% to 8% of cases. When it does there is possibly a 10% chance of the subsequent development of cancer, but vulvectomy is nevertheless justified as a prophylactic measure.

In the remaining majority of cases of chronic epithelial dystrophy there is no special risk of malignant

change and vulvectomy is not justified as a cancer preventive. And as an empirical treatment it does not give satisfactory results even when the skin change appears to be strictly limited and demarcated. Unless the cause is determined and eradicated, epithelial dystrophy recurs in approximately 50% of cases treated by partial or total vulvectomy.

For the searching of records and the follow-up of patients we are indebted mainly to Dr. Winifred Francis; but also to Dr. Mary Mayeur and Dr. R. E. Rewell for details of individual cases. Cases 1 and 3 were under the care of Mr. E. Parry-Jones, to whom we are grateful for permission to report them.

ADDENDUM

Since this paper was completed one more of the 98 women under observation has developed cancer of the vulva, and the circumstances confirm the above conclusions. Histological examination of biopsy specimens of the chronically affected vulval skin was carried out in 1956, and showed epithelial activity of a degree which one of us (A. S. W.) considered disturbing. The lesion was so widespread and ill demarcated clinically as to be unsuitable for excision, so it was treated conservatively with some improvement. In view of the histological findings, however, the patient was observed regularly, and when symptoms relapsed in 1960 further biopsy specimens were examined without any more alarming epithelial changes being found. In April, 1961 (after five years' observation), however, the patient reported that she had had pain in one area of the vulva for one week. This area then had a red appearance, and on section showed cancer *in situ* with possibly early invasion of malignant cells at one point.

REFERENCES

Berkeley, C., and Bonney, V. (1909). *Brit. med. J.*, **2**, 1739.
Bibby, A. V. G. (1957). *J. Obstet. Gynaec. Brit. Emp.*, **64**, 263.
Bonney, V. (1908). *Lancet*, **1**, 1465.
—— (1938). *Proc. roy. Soc. Med.*, **31**, 1057.
Böttger, H., and Dittmann, E. (1957). *Geburtsh. u. Frauenheilk.*, **17**, 1097.
Corscaden, J. A. (1956). *Gynecologic Cancer*, 2nd ed. Williams and Wilkins, Baltimore.
Darier, J. (1928). *Précis de Dermatologie*, 4th ed. Masson, Paris.
Gerrard, E. A. (1932). *Trans. North Engl. obstet. gynaec. Soc.*, p. 65.
Green, T. H., jun., Ulfelder, H., and Meigs, J. V. (1958). *Amer. J. Obstet. Gynec.*, **75**, 834.
Hunt, Elizabeth (1940). *Diseases Affecting the Vulva*, 1st ed. Kimpton, London.
—— (1954). Ibid., 4th ed. Kimpton, London.
Jayle, F. (1906). *Rev. Gynéc.*, **10**, 633.
Jeffcoate, T. N. A. (1949). *Brit. med. J.*, **2**, 1196.
—— (1955). *Med. Wld (Lond.)*, **82**, 136.
—— (1957). *Principles of Gynaecology*. Butterworth, London.
—— Davie, T. B., and Harrison, C. V. (1944). *J. Obstet. Gynaec. Brit. Emp.*, **51**, 377.
Langley, I. I., Hertig, A. T., and Smith, G. Van S. (1951). *Amer. J. Obstet. Gynec.*, **62**, 167.
Lunin, A. B. (1949). Ibid., **57**, 742.
McAdams, A. J., jun., and Kistner, R. W. (1958). *Cancer*, **11**, 740.
Novak, E., and Novak, E. R. (1952). *Textbook of Gynecology*, 4th ed. Williams and Wilkins, Baltimore.
Rewell, R. E. (1960). *Obstetrical and Gynaecological Pathology for Postgraduate Students*. Livingstone, Edinburgh.
Rubin, I. C., and Novak, J. (1956). *Integrated Gynecology: Principles and Practice*, Vol. 2. Blakiston, New York.
Stening, M., and Elliott, P. (1959). *J. Obstet. Gynaec. Brit. Emp.*, **66**, 897.
Stewart, D. B. (1959). *Modern Trends in Gynecology and Obstetrics*—Proc. 2nd int. Congr. Gynec. Obstet., Vol. 1, p. 809. Beauchemin, Montreal.
Taussig, F. J. (1917). *Amer. J. Obstet.*, **76**, 794.
—— (1922). *Surg. Clin. N. Amer.*, **2**, 1559.
—— (1930). *Arch. Derm. Syph. (Chicago)*, **21**, 431.
—— (1931). *Diseases of the Vulva*. Gynecological and Obstetrical Monographs, Vol. XIII. Appleton, New York.
—— (1940). *Amer. J. Obstet. Gynec.*, **40**, 764.
Wallace, H. J. (1955). In *Modern Trends in Obstetrics and Gynaecology*, 2nd ser., ed. K. Bowes, p. 386. Butterworth, London.
—— and Whimster, I. W. (1951). *Brit. J. Derm.*, **63**, 241.
Way, S. (1951). *Malignant Disease of the Female Genital Tract*. Churchill, London.

SYNDROME OF MITRAL INCOMPETENCE, MYOCARDITIS, AND PULMONARY HYPERTENSION IN NIGERIA

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[WITH SPECIAL PLATE]

Rheumatic mitral-valve disease has been studied intensively during the past 25 years. Mitral stenosis is rare in Nigerians, but one of us (Abrahams, 1959) described a peculiar form of mitral-valve disease which is common in West Africa but unusual elsewhere. In this paper is reported a more detailed study of the syndrome (presenting as mitral incompetence and pulmonary hypertension), which may be caused by an unusual form of rheumatic carditis.

Material

The study covered 50 patients with signs of mitral incompetence and pulmonary hypertension observed over a relatively short period; most of them were examined by both authors, and many were admitted to hospital for treatment and more prolonged observation. Electrocardiograms were recorded and a radiological examination was made in all cases. Right heart catheterization was carried out successfully in 15 cases. Necropsy was performed in 13 cases.

All the patients came from the western region of Nigeria. Most of the tribes of the south were represented, but Yorubas, who are the majority people of the western region, predominated. All were young by Western standards, but not in their own community, where the expectation of life is short. At the last official census in 1952 the population of Ibadan Province was over 1½ million, and less than 8% were over the age of 50. The diet of these people consists mainly of carbohydrate and is deficient in protein, but none of the patients was obviously malnourished at the time of examination.

Clinical Features

Women were affected at least twice as frequently as men, and over three-quarters of the series were under 30 years of age. It was not easy to obtain an accurate history, because these people have little sense of time and find it difficult to describe symptoms. However, the majority complained of substernal pain; in some cases this was clearly cardiac pain, but generally it was impossible to distinguish pain from the sense of tightness which accompanied effort dyspnoea. Nocturnal dyspnoea was uncommon, and most patients could lie flat without discomfort. Cough was a constant complaint, but haemoptysis was very rare.

Swelling of the legs and abdomen was the presenting symptom in half the cases, and in some had already

*In receipt of a grant from the Nuffield Foundation for the purpose of this study.

assumed gross proportions. We were unable to obtain a history of preceding acute illness in any patient, and in particular sore throat and arthralgia were conspicuously absent.

The physical signs were those of mitral incompetence and pulmonary hypertension of varying severity, with or without right heart failure. The radial pulse was small in volume, and the average systemic arterial pressure was only 112/76 mm. Hg over the whole series. When it appeared that mitral incompetence was severe the pulse was small and had a collapsing quality. Sinus rhythm was present in all except one patient, who had atrial fibrillation. Ventricular extrasystoles caused pulse irregularity in many patients. The mean jugular venous pressure was always raised when there was oedema with ascites. In a number of patients a large *cv* wave indicated tricuspid incompetence, and some of these subsequently showed evidence of an unusual lesion affecting the right ventricle. When there was severe pulmonary hypertension the *a* wave was increased, but giant *a* waves were not seen.

The praecordium and left chest was deformed by great cardiac enlargement in some of the younger patients. Palpation at the left sternal edge showed increased pulsation from right ventricular hypertension and hypertrophy. Pulsation over the pulmonary artery was palpable in most cases. Some degree of left ventricular hypertrophy could be felt at the apex in the majority, and in those with great mitral incompetence a powerful heave was followed by a diastolic rapid inflow beat.

All patients had an apical pansystolic murmur; in some this was palpable. A mitral diastolic murmur was not generally heard; however, in a few patients in whom active carditis was suspected a short mid-diastolic murmur was heard at some time. A third heart sound was audible in half the cases. The pulmonary second sound was always louder than normal, and in patients with evidence of severe pulmonary hypertension "splitting" was close and there was a pulmonary diastolic murmur in nine. There was no clinical evidence of aortic-valve disease in any of the patients, and this was confirmed by the absence of aortic-valve disease in the 13 cases that came to necropsy.

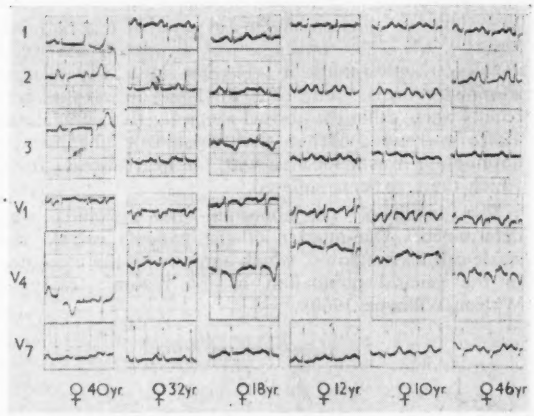
Congestive cardiac failure was present in half the patients when first examined; that these patients had not presented earlier appeared to be due to poverty and ignorance rather than the natural history of the disease. In 16 patients there were signs suggesting an active carditis in addition to those of established mitral incompetence and pulmonary hypertension. These patients had slight fever and tachycardia out of proportion to the rise in temperature; some had a transient mid-diastolic murmur, and others pericardial friction. Six of these patients had a high venous pressure, swelling of the face, and a tender liver without dependent oedema or ascites. Pulmonary oedema developed in two, which was in contrast with the rest of the patients in whom the evidence of selective left heart failure was minimal.

Radiology

The radiological signs were remarkably uniform. Pulmonary venous congestion (hilar congestion) was absent or minimal (Special Plate, Fig. 1). Septal lines were not seen. The peripheral lung fields were clear, as in other kinds of pulmonary hypertension. The main pulmonary arteries were large and pulsated vigorously in some patients, but the branches were attenuated and there was never any suggestion of plethora. In some of the older patients with tricuspid incompetence and very large hearts the pulmonary arteries were less conspicuous (Special Plate, Fig. 2). The aorta was small. The heart shadow was always larger than normal. The right atrium was invariably larger and much more conspicuous than the left, which was never greatly enlarged. However, there was often a left auricular bulge below the prominence of the right ventricular outflow tract and pulmonary artery (Special Plate, Fig. 1). Systolic expansion of the left atrium was seen in a few patients. In advanced cases the great enlargement of the right atrium, the convexity of the upper left border, and the small vascular pedicle gave the heart a globular appearance.

Electrocardiography

Sinus rhythm was usual, but ventricular extrasystoles were common, as in other forms of mitral incompetence. One patient had atrial fibrillation, and another, who was thought to have active carditis, had transient first-degree heart-block. Electrocardiographic signs of right ventricular hypertrophy were present in all patients (Text Fig. 1). Right bundle-branch block occurred



TEXT FIG. 1.—Electrocardiograms from six patients, mounted from left to right in ascending order of right ventricular dominance. Notched and biphasic P waves indicate right and left atrial hypertrophy.

twice, and was also considered to indicate right ventricular hypertrophy. There was additional evidence of left ventricular hypertrophy in most cases. Monophasic R waves were present in V7 in most, and even when evidence of right ventricular hypertrophy was extreme only a shallow S appeared in V7. Large P waves indicated right atrial hypertrophy in all cases, and there was additional evidence of left atrial hypertrophy in the majority. Biphasic P waves in V1 were almost invariable (Text Fig. 1).

Cardiac Catheterization

Cardiac catheterization was performed by the usual technique, but in some instances there was difficulty in entering the right ventricle when the right atrium was greatly enlarged. Even when the right ventricle and pulmonary artery were successfully entered (15 cases) it was not always possible to obtain a satisfactory "wedge" pressure (10 cases). The pulmonary arterial

pressure was abnormally high in each case, and the "wedge" pressure, which was assumed to reflect the left atrial pressure, was also high. Cardiac output was low and the pulmonary vascular resistance was raised to 527, 713, and 541 dynes/sec./cm.[5] in three cases (Nos. 16, 18, and 19) where calculation of this factor was possible. Other results are shown in Table I.

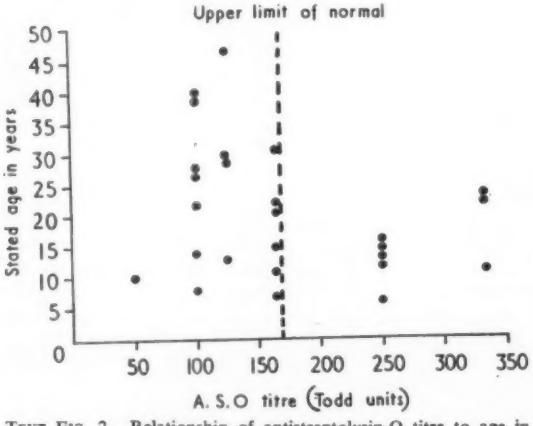
TABLE I.—*Pressures (mm. Hg) Obtained at Catheterization in Right Atrium (R.A.), Ventricle (R.V.), Main Pulmonary Artery (M.P.A.), and Wedged Pulmonary Artery in 15 Patients*

Case No.	Age (Yrs.)	Sex	R.A. Pressure	R.V. Pressure		M.P.A. Pressure		Wedge Pressure
				Absolute	Mean	Absolute	Mean	
2	27	F	+5	60/2	25	62/30	44	19
4	47	F	+4	92/4	46	90/38	61	—
5	8	M	+12	60/5	26	63/22	37	22
6	16	F	0	55/0	27	57/25	40	15
7	16	M	+4	62/4	35	60/35	45	24
9	28	F	+6	60/0	—	60/35	42	—
12	32	F	+12	85/5	43	83/44	58	28
14	14	F	+5	104/0	48	102/42	69	—
15	22	M	+8	70/5	—	68/30	—	20
16	41	F	+2	65/0	31	65/37	45	16
17	25	F	+4	80/0	31	81/38	51	—
18	15	M	+6	75/3	—	75/34	56	23
19	12	F	0	60/0	21	58/25	41	23
20	11	F	+2	100/0	42	98/43	62	28
21	27	M	+4	—	40	—	53	—

Other Investigations

All the patients had anaemia. Half of the men and one-third of the women had haemoglobin levels below 70%, and six children under 10 years of age had the lowest levels. The anaemia was always of the iron-deficiency type, though it was often refractory to oral administration of iron. The total and differential cell counts were within the normal range for this population. There was no evidence that significant unexplained eosinophilia was associated with the heart disease from which these patients suffered.

The incidence of abnormal haemoglobins was determined. One-quarter of the patients carried the sickle-cell haemoglobin, which agrees with the incidence in the general population of the region* (24.4%—Watson-Williams, 1960).



TEXT FIG. 2.—Relationship of antistreptolysin-O titre to age in 27 patients.

The erythrocyte sedimentation rate, measured in a number of patients, was always moderately raised; it seemed to bear no relation to the patient's heart condition or to the progress of the disease when serial estimations were made. As malaria, ancylostomiasis, and other infections are rife in Nigeria the significance of the E.S.R. in our cases could not be assessed.

It is well known that the plasma-protein pattern of Africans is unlike that of Europeans in that the albumin/globulin (A/G) ratio is reversed. Observations were made on only nine of our patients, and the plasma-protein values corresponded to the usual pattern for this population as described by Edozien (1958). The mean value for total proteins was 6.7 g. per 100 ml. The average serum albumin was 2.5 g. and the serum globulin 4.7 g. per 100 ml., giving an average A/G ratio of 1/1.88 (the increase being mainly due to γ-globulin).

Before determining the antistreptolysin-O (A.S.O) titre the local normal was established in 100 Nigerian blood donors of all ages and both sexes, and was found to be the same as in Europeans (upper limit of normal 166 Todd units). The results in 26 patients in the present series are shown in Text Fig. 2. The relation between the onset of symptoms or start of the disease and the level of antibody titre could not be determined owing to the paucity of history. Over 50% (14) of the 26 patients had an A.S.O titre above normal, and in 90% it was over 100 Todd units, whereas only 13% of the controls had an A.S.O titre above normal, and in more than 50% it was below 50 units. Eight patients with a titre of 250 units or more were under 25 years of age. A.S.O titres had been determined in six of the patients who came to necropsy (Table II).

TABLE II.—*Antistreptolysin-O Titre and Necropsy Findings in Six Fatal Cases*

Case No.	Sex and Age	A.S.O Titre	Histology
14	F 15	100	Many Aschoff nodules
15	M 23	100	Aschoff nodules scanty and much fibrosis
24	F 15	250	Many Aschoff nodules
28	F 26	200	Non-specific myocarditis and endocardial thickening
29	F 25	100	Aschoff nodules and endocardial thickening
45	M 7	250	Aschoff nodules scanty and endocardial thickening

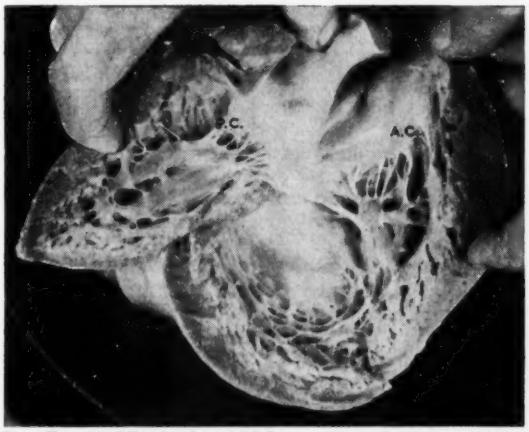
Necropsy Findings

Confirmation of the clinical diagnosis of mitral-valve disease causing mitral incompetence was obtained in the 13 patients who came to necropsy. No other valve was affected. However, examination of these hearts suggested that more than one disease process had led to an identical clinical picture. There was evidence of myocarditis in each heart; in some this was indistinguishable from rheumatic carditis, while in others the reaction appeared to be of a non-specific interstitial variety. Endomyocardial fibrosis of varying degree was present in some hearts irrespective of the kind of inflammatory reaction found histologically. All the patients had died of congestive cardiac failure, and one had recent ulcerative valvulitis.

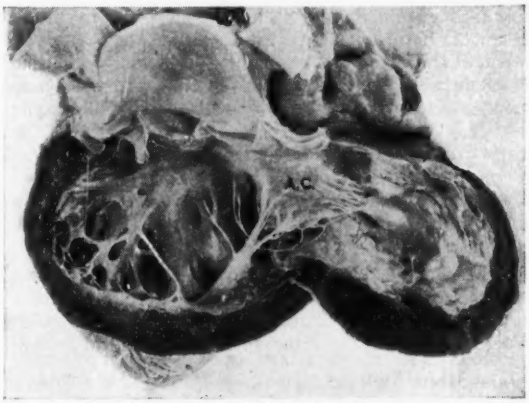
The left atrium was moderately enlarged and its wall was thicker than normal, being up to 5 mm. in one case. This left atrial thickening was mainly due to muscle hypertrophy rather than endocardial hyperplasia, which was, however, present on the posterior wall in two cases. Each heart showed some degree of left ventricular hypertrophy (Text Fig. 3). There was a variable amount of macroscopic endomyocardial fibrosis affecting the inflow tract of the left ventricle (Text Fig. 4) and varying from a slight patchy opacification of the endocardium to a gross silting up of the apical lumen (Text Fig. 5). In the most severely affected hearts the

*Figures taken from an analysis of 5,730 blood donors.

posterior papillary muscle and the valve chordae were incorporated in this process, and the posterior cusp of the valve was then greatly deformed (Text Fig. 5). In all cases the mitral-valve cusps were thickened, and in one case ulcerative endocarditis had arisen on the deformed valve.



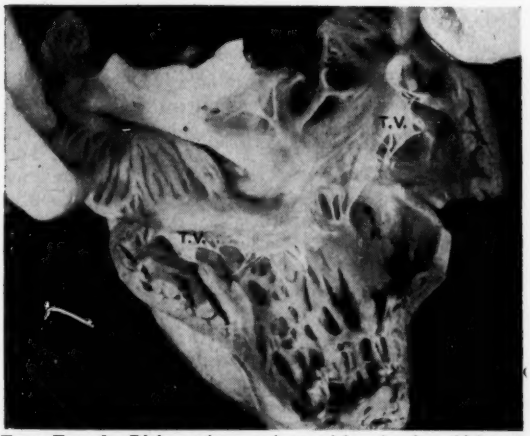
TEXT FIG. 3.—Mitral valve and left ventricle. Chordae tendineae, particularly of posterior papillary muscle, are shortened; hypertrophy of left ventricle with patchy fibrosis. (P.C.=posterior and A.C.=anterior cusp.)



TEXT FIG. 4.—Endomyocardial fibrosis in left ventricle. Posterior papillary muscle is affected in this process; the outflow tract is almost free of endocardial thickening.

TEXT FIG. 5.—Endomyocardial fibrosis of left ventricle. A large thrombus—part calcified—can be seen at apex. Posterior papillary muscle is incorporated in ventricular wall (P.C.) and valve is grossly distorted and fibrotic.

A curious but common finding was an increase in the number of trabeculae carneae, which were thinner than normal and gave the endocardial surface a lace-like appearance. Fresh thrombi and partially organized ones were commonly found in the spaces between the trabeculae. The right ventricle was always thicker than normal, and in some hearts hypertrophy of the trabeculae had greatly diminished the size of the ventricular cavity; in other cases the right ventricular outflow tract was much dilated, producing a large oblique bulge over the anterior surface of the heart (Text Fig. 6).



TEXT FIG. 6.—Right atrium and ventricle, showing thickened trabeculae and dilated outflow tract. (T.V.=tricuspid valve; P=pulmonary valve.)

None of the hearts showed evidence of coronary-artery disease. The diameter of the pulmonary artery was always greater than that of the aorta, which was normal. Macroscopic evidence of previous pericarditis was present in only two cases.

On microscopical examination one or more of the following lesions were found: (*a*) "rheumatic" carditis with Aschoff bodies; (*b*) a non-specific inflammatory myocarditis; (*c*) fibrosis which was maximal in the endomyocardium.

(*a*) The presence of Aschoff bodies was the sole histological criterion for recognition of "rheumatic" carditis. They were typical of the Aschoff body found in the usual form of rheumatic carditis, having characteristic basophil cells with ragged edges and occasional fibroblasts. There was granular collagen between the cells, and scattered round cells occurred around the mass (Special Plate, Fig. 3). The nodes were usually spindle-shaped and adjacent to small blood-vessels (Special Plate, Fig. 4). This reaction was found mainly in the ventricular myocardium and was present in eight of the 13 hearts; in four it was the only abnormality, while in three it was associated with endomyocardial fibrosis, and in one other an intense rheumatic reaction was accompanied by a "non-specific" myocarditis. In contrast to the usual form of rheumatic carditis the mitral valve was only slightly affected. The other valves were normal. Aschoff nodules were also found in the muscle and endocardium of the atria.

(*b*) Three hearts showed a non-specific myocarditis which was focal and interstitial, with infiltration by eosinophil and mononuclear cells.

(*c*) Some degree of endomyocardial fibrosis extending for a varying depth into the left ventricle was present in three hearts with no other histological abnormalities; it was associated with scanty Aschoff-nodule formation in three hearts and with non-specific myocarditis in two others.

Histological examination of the lungs showed only slight evidence of vascular hypertrophy; most small arteries and arterioles were normal. Furthermore, the vascular pattern shown by post-mortem pulmonary angiography was normal in the case of a 15-year-old girl whose pulmonary arterial pressure was 102/42 mm. Hg (Case 14) one year before death.

Discussion

The constant clinical findings of a mitral pansystolic murmur, an overactive left ventricular apex-beat, a loud pulmonary second sound, and palpable pulmonary arterial pulsation, together with the radiological and electrocardiographic signs, clearly indicated the presence of mitral incompetence and pulmonary hypertension in these patients. Confirmatory evidence was obtained from cardiac catheterization, and the necropsies showed that there was severe myocardial disease in addition to organic mitral valvulitis and pulmonary hypertension. This syndrome appears to be common in Nigeria, in contrast to its rarity in Great Britain. Two features must be kept distinct in considering the pathogenesis of the condition: one concerns the aetiology of the cardiomyopathy, and the other the mechanism of the pulmonary hypertension and its association with mitral incompetence.

There are many causes of heart-muscle disease, and it is probable that at least two, and perhaps more, are concerned in this disease. Aschoff nodules in the myocardium and rather high antistreptolysin-O titres suggest that there was a form of rheumatic carditis in some cases, though rheumatic fever is very uncommon in Nigeria. It is rare for these people to complain of a sore throat, but it is possible that the haemolytic streptococcus may gain entry by an entirely different route, such as the common tropical leg ulcer. Chorea is rare and arthralgia is equally uncommon, but those affected would not complain unless there was extreme disability. In any case, we might expect that the rheumatic reaction would be different in people suffering from chronic anaemia and chronic infection, and in whom the globulin pattern of the plasma proteins is quite different from that of Europeans. Heart-muscle disease is certainly very common in West Africans, and it may be that the myocardium, having suffered from the effects of anaemia and chronic infection from birth, may be more vulnerable than the endothelium of valves or the soft tissues of joints.

Necropsy on 13 patients in the series showed that a significant proportion had some degree of endomyocardial fibrosis. It is not suggested that this is caused by rheumatic carditis, but it appears that cardiomyopathy, whether due to inflammation, nutritional deficiency, or other metabolic disorder, may during an acute phase precipitate mural intratrabecular clots, which subsequently become organized. This process may be recurrent and lead to endomyocardial fibrosis.

There have been many accounts of cardiomyopathy in West Africans since Bedford and Konstam (1946) first drew attention to this condition. Davies (1948) described an apparently similar form of heart disease in Uganda, calling it endomyocardial fibrosis, and subsequent papers from Uganda have extended the original observations (Ball *et al.*, 1954; Williams *et al.*, 1954). Endomyocardial fibrosis has now been reported from many parts of the world, including the Sudan (O'Brien, 1954), Nigeria (Nwokolo, 1955), Rhodesia (Gelfand, 1958), Ceylon (Nagaratnam and Dissanayake, 1959), Malaya (La Brooy, 1957), Hawaii (Stemmermann, 1957), America (Fienberg and Holzman, 1951; Clark *et al.*, 1956; Fisher and Davis, 1958), Great Britain (Gray, 1951; Lynch and Watt, 1957; Evans, 1957), and Holland (Van Buchem *et al.*, 1959). Most of these reports have been concerned with morbid anatomy and theoretical discussion of aetiology. Recently one of us* has seen the syndrome of mitral incompetence and pulmonary hypertension in the French Congo at Brazzaville, where it is common (Peuchot, 1959), and in Uganda, where both clinical cases and necropsy specimens were seen. Some of these cases were identical with those described here. Patients with the same condition were also seen in Khartum and Accra, but none was seen in South Africa or the Rhodesias (Baldachin, 1959); the cardiovascular collagenosis with endocardial thrombosis described by Becker and his colleagues (1953) is different.

The aetiology of endomyocardial fibrosis remains obscure. Infection, malnutrition, avitaminosis, parasitic infestation, and allergy have all been discussed in the papers mentioned. In the present series endomyocardial fibrosis of the left ventricle was clinically indistinguishable from rheumatic mitral incompetence, and in several instances both coexisted in the same heart. It is debatable whether this association is casual or causal. The possibility that endomyocardial fibrosis and rheumatic heart disease are related is now being considered in Uganda (Shaper and Williams, 1960). It seems probable that endomyocardial fibrosis is the "final common path" resulting from many different forms of myocardial damage. In Southern Nigeria we suggest that rheumatic carditis may be concerned with this process in some cases.

The dominance of mitral incompetence rather than stenosis is interesting. An analysis of patients with mitral-valve disease attending the hospital at Ibadan (Table III) shows that a combination of mitral incompetence and pulmonary hypertension is common, in contrast to experience in the United Kingdom, where pure mitral incompetence is uncommon and rarely associated with pulmonary hypertension (Brigden and Leatham, 1953). Several factors, including valvulitis, endomyocardial fibrosis, and diffuse myocardial disease, are responsible for this dominance of mitral incompetence. Furthermore, it is probable that in some cases the myocarditis combined with so many unfavourable factors, including continued physical activity, results in death before stenosis can occur.

TABLE III.—*Analysis of Mitral-valve Disease in Out-patients (April–September, 1959)*

Lesion	No. of Cases
Pure mitral stenosis (with or without pulmonary hypertension)	4
Pure mitral incompetence (without any evidence of pulmonary hypertension)	2
Mitral stenosis and incompetence	10
Combined mitral and aortic valve diseases	2
Mitral incompetence with pulmonary hypertension	43
Total	61

There were clinical, electrocardiographic, and radiological signs of pulmonary hypertension in all patients in this series. High pulmonary arterial pressures were

*D. G. A. (with a grant from the Rockefeller Foundation).

confirmed by cardiac catheterization. There was evidence of chronic elevation of left atrial pressure apart from the raised " wedge " pressures obtained at catheterization. The electrocardiogram showed left atrial hypertrophy as in mitral stenosis, and at necropsy there was great atrial hypertrophy without dilatation in some cases. Severe left atrial hypertension is in contrast to the simple systolic type of hypertension which exists in pure rheumatic mitral incompetence in Europeans. In these patients the ventricular diastolic inflow may be impeded by stiffness of the hypertrophied myocardium associated, in some cases, with the occlusive and splinting effects of endomyocardial fibrosis. It is therefore probable that the pulmonary hypertension is, as in many cases of mitral stenosis, due to the passive effects of the high left atrial pressure coupled with a vasoconstrictive element at a precapillary level. It remains to mention the possibility that pulmonary vasoconstriction is an excessive response to the pulmonary venous hypertension because of chronic anaemia or some other unknown factor. Left atrial hypertrophy, in contrast to dilatation, parallels the absence of atrial fibrillation. It is probable that atrial systole is an essential component in the maintenance of a cardiac output sufficient to sustain life in these patients.

Finally, we wish to stress the importance of the soil in which this form of heart disease thrives. Anaemia is usual; intestinal parasitism is rampant; in addition malaria is endemic, and the dysenteries, both bacterial and protozoal, are widespread. Protein malnutrition is rife, especially in infants and children. In the presence of persistent chronic ill-health of this nature it is difficult to believe that the myocardium remains unaffected and healthy. It is probable that anaemia alone, when severe and protracted, may lead to cardiomyopathy (Somers, 1959). On top of all this it must be emphasized that these patients are always ambulant, and never rest unless it is physically impossible for them to stand and walk. This continued physical activity may have further adverse effects upon an already damaged myocardium.

Summary

The clinical, radiological, and electrocardiographic features of 50 Nigerian patients who presented with heart failure due to mitral incompetence and pulmonary hypertension have been investigated. Necropsy examination of 13 cases showed that there was severe left ventricular myocardial disease and mitral-valve disease. Most hearts showed some degree of macroscopic endomyocardial fibrosis, and histological examination revealed either a diffuse non-specific inflammatory reaction or a myocarditis with Aschoff nodules which was indistinguishable from rheumatic carditis.

It is suggested that the peculiar pattern of this disease, comprising left ventricular myopathy, mitral valvulitis, and pulmonary hypertension, may be determined not only by its primary cause, but also by the background of chronic anaemia, protein malnutrition, and chronic infestation which is present in these patients and in the population from which they are drawn.

REFERENCES

Abrahams, D. G. (1959). *Lancet*, **2**, 111.
Baldachin, B. J. (1959). *E. Afr. med. J.*, **36**, 542.
Ball, J. D., Williams, A. W., and Davies, J. N. P. (1954). *Lancet*, **1**, 1049.
Becker, B. J. P., Chatgidakis, C. B., and van Lingen, B. (1953). *Circulation*, **7**, 345.
Bedford, D. E., and Konstam, G. L. S. (1946). *Brit. Heart J.*, **8**, 236.
Brigden, W., and Leatham, A. (1953). Ibid., **15**, 55.
Clark, G. M., Valentine, E., and Blount, S. G., jun. (1956). *New Engl. J. Med.*, **254**, 349.
Davies, J. N. P. (1948). *E. Afr. med. J.*, **25**, 10.
Edozien, J. C. (1958). *W. Afr. med. J.*, **7**, 121.
Evans, B. (1957). *Brit. Heart J.*, **19**, 164.
Fienberg, R., and Holzman, D. (1951). *Bull. int. Ass. med. Mus.*, **32**, 34.
Fisher, E. R., and Davis, E. R. (1958). *Amer. Heart J.*, **56**, 537.
Gelfand, M. (1958). *Cent. Afr. J. Med.*, **4**, 365.
Gray, I. R. (1951). *Brit. Heart J.*, **13**, 387.
La Brooy, E. B. (1957). *Proc. Alumni Ass. Malaya*, **10**, 303.
Lynch, J. B., and Watt, J. (1957). *Brit. Heart J.*, **19**, 173.
Nagaratnam, N., and Dissanayake, R. V. P. (1959). Ibid., **21**, 167.
Nwokolo, C. (1955). *W. Afr. med. J.*, **4**, 103.
O'Brien, W. (1954). *Brit. med. J.*, **2**, 899.
Peuchot, G. (1959). Personal communication.
Shaper, A. G., and Williams, A. W. (1960). *Trans. roy. Soc. trop. Med. Hyg.*, **54**, 12.
Somers, K. (1959). *Circulation*, **19**, 672.
Stemmermann, G. N. (1957). *Amer. J. Med.*, **22**, 142.
Van Buchem, F. S. P., Arends, A., and Schröder, E. A. (1959). *Brit. Heart J.*, **21**, 229.
Watson-Williams, E. J. (1960). Personal communication.
Williams, A. W., Ball, J. D., and Davies, J. N. P. (1954). *Trans. roy. Soc. trop. Med. Hyg.*, **48**, 290.

ADRENOCORTICAL STEROIDS IN INTERMITTENT HEART-BLOCK

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Stokes–Adams attacks, by their dramatic nature alarming to the patient and his relatives, are always a matter of grave concern when the usual treatment with sympathomimetic drugs and atropine fails. Until recently there was no other effective simple treatment. There is now, however, growing evidence of the value of adrenocortical steroid therapy in this syndrome.

Prinzmetal and Kennamer (1954), in a paper devoted to treatment of various cardiac arrhythmias, described, almost as a curiosity, the response to corticotrophin of a patient with complete heart-block and Stokes–Adams attacks. A man of 60, after an acute posterior cardiac infarct, had been having repeated and prolonged attacks of syncope for a week with no relief from ephedrine or adrenaline. As a last resort, 40 units of corticotrophin was given: the attacks ceased and normal sinus rhythm returned in a few hours. Relapse a few days later again responded almost immediately, and after 10 days' maintenance treatment heart-block did not recur. Phelps and Lindsay (1957) described a very similar case wherein intramuscular cortisone was effective.

Since then, patients with Stokes–Adams attacks of long standing, associated with chronic heart disease, have been successfully treated with corticotrophin and adrenocortical steroids.

Litchfield *et al.* (1958), using corticotrophin, obtained immediate and permanent freedom from attacks in a man of 35 with ankylosing spondylitis who developed right bundle-branch block and intermittent complete heart-block. The attacks had occurred for nine months, continuing despite ephedrine and frequent injections of adrenaline during six weeks in hospital. A second patient, a man of 45 with rheumatic aortic valvular disease and paroxysmal complete heart-block, had

suffered from Stokes–Adams attacks for five months. They ceased immediately on treatment with corticotrophin, 120 mg. daily, and for five months could be prevented by maintenance dosage above a critical level. Their third patient, a woman of 35 apparently suffering from an acute carditis with a varying degree of atrioventricular block, had had increasingly severe Stokes–Adams attacks for two months. Within six hours of a first injection of corticotrophin complete heart-block was replaced by 2:1 heart-block and in 48 hours atrioventricular conduction was normal.

Caramelli and Tellini (1960) gave prednisone to 31 patients with heart-block, irrespective of its aetiology or degree, and reported the electrocardiographic changes. Conduction returned to normal in five out of seven patients with intermittent complete block, in both of two patients with 2:1 heart-block, and in seven out of ten patients with an abnormally prolonged P–R interval. The rhythm was unaffected in 10 patients with established complete heart-block. Positive results, when obtained, always appeared rapidly, and they thought it pointless to persevere with treatment if there was no effect within four to five days.

Friedberg *et al.* (1960) reported six patients with chronic ischaemic heart disease, in all of whom Stokes–Adams attacks ceased within 48 hours of starting treatment with prednisone, and thereafter could be prevented by an adequate maintenance dose. In all, standard treatment had proved ineffective or at best of transient benefit. Relief of attacks in five out of the six patients was accompanied by a return of normal conduction in the electrocardiogram.

We report here our experience of six patients with Stokes–Adams attacks treated with oral prednisolone.

Case 1

An estate manager aged 72 began, in August, 1959, to suffer from attacks of faintness three to eight times a day. After a momentary disagreeable sensation in the head he would fall to the ground, but was able to get up again immediately. He was unconscious on one occasion only. Sometimes he was able to avoid falling by sitting down. No colour change had been noticed by witnesses. He denied any chest pain or shortness of breath.

He was admitted to hospital some six weeks after the onset. There were no abnormal physical signs except that his heart rate alternated between 40 and 70, with occasional periods of pulselessness lasting for five seconds and accompanied by faintness. An electrocardiogram showed predominant sinus rhythm with a normal P–R interval, right bundle-branch block, and periods of complete atrioventricular block. The attacks continued for a week unaffected by sublingual isoprenaline, 20 mg. four times a day.

Prednisolone, 80 mg. a day, was then begun. He had his last attack 36 hours later, and after two days' treatment his electrocardiogram showed sinus rhythm. After nine days the dose of prednisolone was reduced in stages and he returned home. He has remained entirely well for the past year on a maintenance dose of 10 mg. a day. His electrocardiogram is unchanged, the right bundle-branch block persisting.

Case 2

A retired gardener of 68 began, in October, 1957, to suffer from attacks of loss of consciousness, gradually becoming more frequent until he was averaging four a day. He also experienced frequent feelings of "dizziness" without unconsciousness.

He was admitted to hospital six months after the onset, and typical Stokes–Adams attacks lasting up to 45 seconds, with unconsciousness and twitching and followed by flushing, were repeatedly observed. He had free aortic incompetence with moderate left ventricular enlargement and a blood-pressure of 200/65 mm. Hg. His blood Wassermann reaction was negative. An electrocardiogram showed a P–R interval of 0.24 second and right bundle-branch block.

Attacks averaged five a day for the first 10 days after admission despite ephedrine and adrenaline. Prednisolone, 80 mg. daily, was then started. In the next 24 hours he had about 40 attacks, the next day three, and thereafter none. An electrocardiogram nine days from the starting of treatment showed a P–R interval of 0.2 second and right bundle-branch block. When he had been free from attacks for three weeks he returned home, then receiving prednisolone, 20 mg. a day. Two months later the right bundle-branch block pattern had disappeared. Steroids were given in decreasing doses for a further five months and then stopped.

Three months later syncopal attacks recurred, but these again ceased when he took prednisolone 15 mg. daily. He remained well for a further 18 months, and then was admitted to another hospital with left ventricular failure, complete heart block, and right bundle-branch block, and died next day.

Case 3

A woman of 82 had a series of syncopal attacks, many times a day, for several weeks in 1955. Her doctor noted her pulse rate at times to be below 40. At one time, when the attacks were particularly frequent, she had a temporary left hemiparesis. She was then well for two years, when attacks returned at intervals of a few days. She usually had a brief warning, and then became pale and pulseless, sometimes with involuntary movements. On examination her heart was in regular rhythm at a rate of 90, the left ventricle was moderately enlarged, and her blood-pressure was 210/110 mm. Hg. She had an aortic systolic murmur conducted to the neck and a widely split pulmonary second sound. Her cardiogram showed right bundle-branch block and a P–R interval of 0.2 second. She was treated for 15 months with ephedrine and adrenaline with some temporary improvement.

In July, 1958, she was having three or four attacks a day, usually consisting of "dizziness" only, but with loss of consciousness in about one in ten.

Prednisolone, 80 mg. a day, was then begun. In the first three days she had three attacks, none, and one attack respectively, and thereafter they ceased. The prednisolone was gradually reduced to 15 mg., and she has remained well for over two years.

Case 4

A woman of 58, a high-grade mental defective, complained in 1956 of occasional feelings of faintness, and her pulse was said to have been irregular. Clinically her heart was normal. An electrocardiogram showed non-specific T-wave changes, thought to indicate degenerative heart disease, with normal conduction. On the supposition that her sensations were due to a paroxysmal tachycardia, quinidine, 5 gr. (0.32 g.) t.d.s., was advised. After a few doses her doctor noticed that her pulse rate had fallen to 40, and the drug was abandoned.

She was next seen and admitted in August, 1958, apparently *in extremis*, having suffered for three days from attacks of unconsciousness and twitching every few minutes. She was pale and sweating, and typical Stokes–Adams attacks lasting 15 to 45 seconds were observed every two to three minutes. Her pulse was irregular at about 30 between the periods of standstill. Dissociated atrial waves could be seen in her jugular pulse. Her electrocardiogram showed complete heart-block. There was no improvement with adrenaline.

Hydrocortisone hemisuccinate, 100 mg., was given intravenously and repeated in six hours; and prednisolone,

80 mg. a day, was started by mouth when she could swallow. Fifteen hours after the starting of treatment she was in sinus rhythm with a normal P–R interval, the attacks had ceased, and the improvement in her general condition was dramatic.

After a week the prednisolone was reduced in steps to a maintenance dose of 10 mg. daily, and eventually discontinued in December, 1958. She has remained well for two years, apart from occasional mild "giddiness" of doubtful significance.

Case 5

A lorry-driver of 55 was admitted to hospital in May, 1959, with an acute anteroseptal cardiac infarct. He was severely ill, and during the first 10 days had a number of attacks of paroxysmal tachycardia for which he was given quinidine and later digoxin. These attacks ceased, but were replaced by periods of ventricular asystole, his electrocardiogram showing a P–R interval of 0.24 second and right bundle-branch block. Twelve days after admission he was having Stokes–Adams attacks every five to ten minutes, beginning with olfactory and auditory hallucinations followed by confusion and flushing without loss of consciousness. Digoxin was stopped, but the attacks continued unabated for three days.

Prednisolone, 40 mg. a day, was started, and the attacks ceased within 24 hours. His electrocardiogram then showed normal A–V conduction and right bundle-branch block.

He was discharged home after six weeks in hospital, but was able to live only a very restricted life owing to breathlessness and effort angina. A maintenance dose of prednisolone was continued for a year and then stopped. Three weeks later he died suddenly.

Case 6

A widow of 77 was admitted to hospital in August, 1960, with a six-weeks history of increasingly frequent attacks of loss of consciousness. Bradycardia had been noted for six months. She was moderately short of breath, and during the week before admission had been given an oral diuretic but no digitalis.

On admission she was in mild congestive heart failure with moderate left ventricular enlargement, a ventricular rate of 33, and easily audible dissociated atrial sounds. About 10 typical Stokes–Adams attacks were observed in the first 24 hours in hospital, and there was no response to adrenaline.

Prednisolone, 80 mg. daily, was then begun. About 16 attacks occurred in the next 36 hours and then they ceased. Complete heart-block persisted and there was no significant change in her electrocardiogram. After three weeks in hospital she was returned home on prednisolone, 15 mg. daily, and so far has remained free from attacks for three months.

Discussion

Six patients with heart-block and frequent Stokes–Adams attacks have been treated with prednisolone after adrenaline and related drugs had proved ineffective. In all of them the attacks ceased within 48 hours, and, apart from one patient with established complete heart-block, regular sinus rhythm returned. The clinical improvement was reflected in the electrocardiograms of four patients, with the disappearance of paroxysmal complete block in two and the return to normal of a prolonged P–R interval in two others.

It is well known that patients with Stokes–Adams syncope may improve spontaneously at any time, and that extraneous factors may modify the frequency of attacks. Thus in Case 5 spontaneous recovery was very likely, both because the heart-block appeared 10 days after the onset of cardiac infarction and because it may have been precipitated by digitalis. In Case 6 the attacks became more frequent while the patient was receiving an oral diuretic, and though her serum potassium was normal on admission the possibility of some electrolyte disturbance cannot be excluded. In Cases 1, 2, 3, and 6 Stokes–Adams attacks had been recurring for weeks or months without relief by adrenaline, ephedrine, or isoprenaline, and their prompt cessation can be attributed to prednisolone with some confidence. In Case 4 the duration was a mere three days, but attacks were recurring so often that death seemed imminent, and steroid therapy appeared to be life-saving.

Three possible mechanisms for the effectiveness of corticotrophin or adrenocortical steroid therapy have been suggested.

Prinzmetal and Kennamer (1954) used corticotrophin in the belief that it would suppress inflammation involving the conducting system at the periphery of a cardiac infarct. If this assumption is correct, there is a logical indication for steroid therapy when heart-block complicates myocardial infarction, as there might be if it appears in patients with acute carditis.

Lown *et al.* (1955) presented evidence that cortisone accelerates atrioventricular conduction in the normal heart. They observed that, compared with normal controls, patients with Addison's disease had a significantly prolonged P–R interval in the electrocardiogram, 20% showing first-degree heart-block at some stage. In most cases the P–R interval was shortened by treatment with cortisone. These effects did not appear to result from electrolyte disturbance, for neither in Addisonian crisis nor on treatment with a mineralocorticoid such as deoxycortone was conduction affected. Conversely, the electrocardiograms of patients with Cushing's syndrome tend to show a shortened P–R interval, sometimes of sufficient degree to resemble Wolff–Parkinson–White syndrome. Adrenalectomy is usually followed by a return towards normal. Subsequent writers were aware of these observations, though Phelps and Lindsay (1957) and Litchfield *et al.* (1958) used cortisone and corticotrophin primarily for their anti-inflammatory effect.

Friedberg *et al.* (1960) pointed out that in their patients syncopal attacks from ventricular standstill might cease before there was any change in the basic rhythm, and, indeed, heart-block might later recur and even progress in degree during clinically effective maintenance treatment. They ascribed this to a favourable effect of prednisone on the arousal and stabilization of ventricular pacemakers. Such a mechanism must be assumed for the effect of prednisolone in Case 6, who had permanent complete heart-block.

Our dosage has been empirical, and the optimum must vary from patient to patient and at different stages of the disease. Caramelli and Tellini (1960) found that 40 mg. of prednisone a day was required to produce a full response where one could be obtained, and in their patients larger doses showed no advantage.

Maintenance treatment for a long period is likely to be needed by patients with chronic heart disease, and we have found that 10 to 15 mg. of prednisolone daily suffices. In Case 4 it was stopped after five months without a return of attacks. When maintenance prednisolone was stopped after seven months, Case 2 relapsed, but the attacks again responded to 15 mg. daily. In one of the patients reported by Litchfield *et al.* (1958), 60 mg. of prednisone or 200 mg. of

cortisone was insufficient to control attacks, although corticotrophin, 50 mg. daily, was effective.

Steroid therapy has so far proved safe in these patients. The usual accepted contraindications were absent. It is, however, interesting that, in Case 2, Stokes–Adams attacks occurred with greater frequency during the first day after starting treatment. Such a phenomenon has not been noted before. There is a theoretical possibility that steroids might interfere with the process of healing after cardiac infarction, but this does not seem to be the case in experimental infarcts in laboratory animals. Furthermore, in such cases treatment for a few days only may be enough.

Summary

Six patients with heart-block and Stokes–Adams attacks have been treated with prednisolone after the failure of conventional measures, and in all six the attacks ceased within 48 hours. Subsequently all patients have remained free from episodes of syncope for a long period.

Similar results have been reported by others, and treatment with adrenocortical steroids appears often to be useful when sympathomimetic drugs are ineffective. In general, a favourable response is most likely when Stokes–Adams attacks occur in a patient with atrio-ventricular block which is unstable. In such cases a return to normal rhythm can be hoped for. Once complete heart-block is established, the rhythm is usually unaffected by steroid treatment, but if it is complicated by syncopal attacks from ventricular stand-still, steroid therapy may still relieve them. Prolonged maintenance treatment is suggested in patients with chronic ischaemic heart disease.

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REFERENCES

Caramelli, Z., and Tellini, R. R. (1960). *Amer. J. Cardiol.*, **5**, 263.
Friedberg, C. K., Kahn, M., Scheuer, J., Bleifer, S., and Dack, S. (1960). *J. Amer. med. Ass.*, **172**, 1146.
Litchfield, J. W., Manley, K. A., and Polak, A. (1958). *Lancet*, **1**, 935.
Lown, B., Arons, W. L., Ganong, W. F., Vazifdar, J. P., Levine, S. A. (1955). *Amer. Heart J.*, **50**, 760.
Phelps, M. D., jun., and Lindsay, J. D., jun. (1957). *New Engl. J. Med.*, **256**, 204.
Prinzmetal, M., and Kennamer, R. (1954). *J. Amer. med. Ass.*, **154**, 1049.

Dr. J. A. SCOTT, the London County Council's Medical Officer of Health, reports that the number of emergency calls received by the London Ambulance Service increased by more than 10,000 between 1958 and 1960. In 1958 the number of calls was 99,188; in 1959 104,983; in 1960 109,551. Despite the extra journeys the average time taken by ambulances to reach the scenes of accidents was only a fraction of a minute longer than in previous years—6.5 minutes in 1958, 6.8 minutes in 1960. On average, patients were admitted to hospital 21.6 minutes after the calls for help were received. As usual, women about to give birth outnumbered all other kinds of patients (25.2% of the total). Street accident cases came next (20%), displacing from second position cases of illness in public places. There were 22,709 patients who had been involved in street accidents, 1,000 more than in 1959. The number of patients who had been victims of assault rose by nearly 700 to 4,107.

HYPERBILIRUBINAEMIA AND PERCEPTIVE DEAFNESS

BY

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The association between kernicterus and perceptive deafness has been well known since Crabtree and Gerrard (1950) demonstrated that deafness was present in 16 out of 20 cases of athetoid cerebral palsy.

After the identification by Claireaux, Cole, and Lathe (1953) of bilirubin as the pigment in the brain cells in kernicterus, evidence accumulated to incriminate bilirubin as the principal factor in the causation of neurological complications in haemolytic disease of the newborn. This evidence has been reviewed by Lathe, Claireaux, and Norman (1958) and by many others, and hardly needs to be recapitulated. It seemed of importance, therefore, to ascertain by a prospective type of investigation how often perceptive deafness might occur as the sole result of hyperbilirubinaemia and whether this could occur at a bilirubin level lower than necessary to cause athetoid cerebral palsy.

Material

The children selected for this study were infants born at Queen Charlotte's Maternity Hospital between the end of 1953 and the middle of 1957 who suffered from hyperbilirubinaemia.

From the middle of 1954 at Queen Charlotte's Hospital regular daily estimations of total bile pigments and conjugated bilirubin have been performed on all babies showing any sign of jaundice at all, and are repeated on each baby until the bilirubin level begins to fall.

An adaptation of the Evelyn and Malloy technique, as described by Lathe and Ruthven (1958), has been used, but at the beginning of 1957 the factor employed in calculating the bilirubin was altered, as it was found that the readings had been too high by about 20%. The figures given in this paper have therefore been adjusted to make them comparable to the more recent estimations. Accordingly, out of 107 infants, after adjustments as mentioned above, it was found that only 20 reached a bilirubin level of 20 mg./100 ml.

For the purposes of this investigation it was possible to examine 50 children completely. Full assessment of their hearing was carried out, and this study of the relationship of hyperbilirubinaemia to perceptive deafness is based on the special investigation of these 50 children. In this group were 13 with a bilirubin concentration of over 20 mg./100 ml.

Results of Hearing-tests

All these children had a detailed audiological examination, and perfectly normal hearing was found in 41 out of 50. In three there was a significant impairment of hearing of conductive type, but this is not relevant in this context.

In six children bilateral perceptive partial deafness was detected. As seen from the audiographs (see Chart), the

pattern of hearing loss is similar in all but one of the cases. Hearing is either within normal limits or only slightly impaired in the lower range of notes, with a gradually increasing impairment in the higher range. Often the hearing for the highest notes is a little better, with a maximum degree of impairment in the range of 2,000 to 4,000 c/s. This type of hearing loss is characteristic for deafness from haemolytic disease as found in previous studies (Fisch, 1957). Severe deafness, as shown in one of the cases in this series, is comparatively rare. In three of the six children perceptive deafness had been diagnosed before this investigation and appropriate measures were taken for training and rehabilitation at an early age.

In one case deafness was noticed before, but its true nature had not been recognized, probably because of a superimposed conductive hearing loss.

In two cases hearing loss was not suspected at all before this investigation, although in one of these there was marked articulation defect. The other child was seemingly backward, but later it was realized that her I.Q. had been assessed as being low because deafness was not suspected, and consequently the appropriate method for testing intelligence had not been applied.

Clinical Condition and Bilirubin Levels in the Partially Deaf Children

Of the six children suffering from perceptive deafness, two were typical examples of kernicterus resulting from Rh iso-immunization (Cases A and C, see Table), and had a marked athetoid cerebral palsy. Their maximum serum bilirubin concentration was respectively 34 and 30 mg./100 ml.

Details of Children With Perceptive Deafness

Case	Clinical	Max. Bilirubin mg./100 ml.	Synkavit mg.	Birth Wt. lb. oz.	Birth Wt. g.	Maturity in Weeks	Blood
A	Kernicterus.						
	Deafness	34	70	7 12	3,545	37	Rh inc.
B	Deafness	26	15	2 11	1,200	31	No immun.
C	Kernicterus.						
	Deafness	30	Nil	7	3,175	41	Rh inc.
D	Deafness	19·0	50	5 11	2,580	34	No immun.
E	,,	20·3	20	6 14	3,120	38	Anti-A haemolysins present
F	Kernicterus.						
	Deafness	15	30	1 8	680	? 31	No immun.

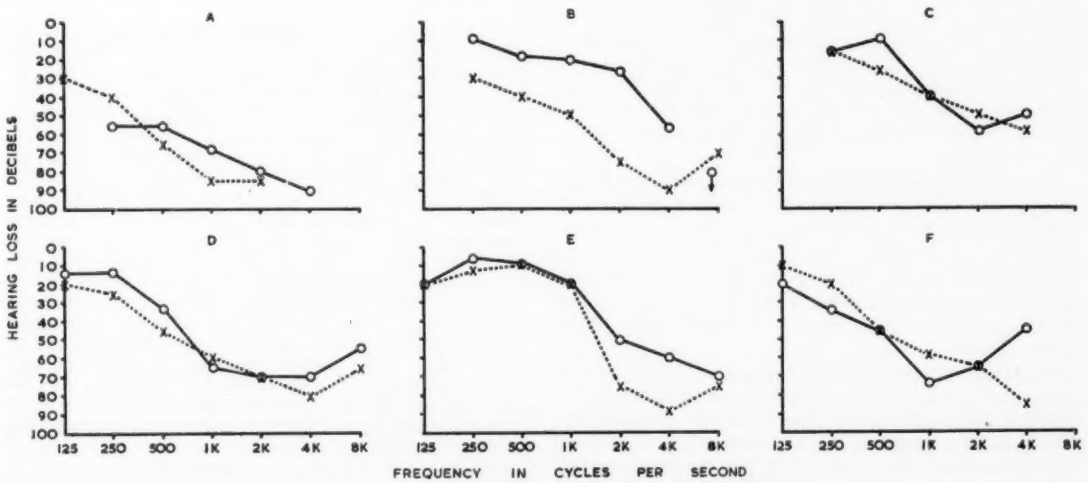
The mother of another (Case E) had alpha-haemolysins in her blood at a low titre, and it is reasonable to assume an A–O blood-group incompatibility as the probable cause of the symptoms.

Three children were premature, with no findings to suggest iso-immunization. One of these (Case F) developed kernicterus and cerebral palsy. The remaining two (Cases B and D) appeared physically normal, although one of us during the examination of hearing, when various tests based on play technique were carried out, observed that all these children had a very mild muscular incoordination in their hands and other parts of the body. They were also regarded by their parents as children who were "restless," "fidgety," and "could not keep still."

It can be seen from the Table that the maximum bilirubin concentration in Cases B, D, E, and F was not very high and would be regarded by many as being well within the range of safety. It was therefore necessary to consider whether any other factors existed which could potentiate the effects of bilirubin.

It was found that all but one (Case C) of the children had received vitamin K in the form of "synkavit." The total dose varied from 15 to 70 mg. in daily amounts of 5 to 20 mg. for the first three to five days. For example Case B, a premature child of 31 weeks' gestation and weighing 2 lb. 11 oz. (1,200 g.) at birth, was given 10 mg. daily for the first three days, and for some odd reason the same amount on the sixth and seventh days. His serum bilirubin rose to 19 mg. on the fifth day. Case E, of 38 weeks' gestation and 6 lb. 14 oz. (3,120 g.) at birth, had a bilirubin concentration of 20.3 mg. on the third day and received 10 mg. of synkavit on the first and third days. Case F, of allegedly 31 weeks' gestation and weighing only 1 lb. 8 oz. (680 g.) at birth, was given 10 mg. of synkavit daily over the first three days of life. The maximum serum bilirubin level was 15 mg. on the sixth day of life.

These babies had therefore all received vitamin K analogue in a high dosage.



Audiographs of six children with perceptive deafness.

Discussion

A prospective study of children who showed signs of jaundice during the neonatal period revealed six cases with congenital perceptive partial deafness. In two of these children the concentration of bilirubin was very high, and both developed kernicterus, athetoid cerebral palsy, and deafness. In four the bilirubin concentration was relatively not high (regarded by many as within the margin of safety). Three of them did not develop clinically detectable kernicterus, had no cerebral palsy, but were affected by perceptive deafness of the type which is known to be associated with haemolytic disease of the newborn. These children received comparatively high doses of synkavit.

Does it mean that the bilirubin concentration regarded as being within the margin of safety (15 mg., 19 mg., 20.3 mg., and 26 mg./100 ml. in these four cases) can still cause a perceptive hearing loss without producing other marked clinical symptoms or signs? Or does it mean that other contributing factors produced the damage resulting in hearing loss?

A possibility that vitamin K in the presence of only a moderate degree of hyperbilirubinaemia was responsible for the perceptive deafness cannot be ignored. This may not be the only contributing factor, because several babies had bilirubin concentrations of about the same levels and had also received vitamin K in a similar dosage but did not show any evidence of deafness. On the other hand, the possibility that perceptive deafness in the three cases with a relatively low maximum bilirubin concentration was associated with vitamin K therapy is supported by the fact that no further instances of deafness occurred after the cessation, at the end of 1954, of the habit of giving vitamin K in doses of more than 2 mg.

It is accepted that kernicterus is usually the result of hyperbilirubinaemia (Lathe *et al.*, 1958) and also that kernicterus is more likely to occur in premature infants if vitamin K is given in excessive amounts (Allison, 1955; Laurance, 1955).

The relatively low bilirubin concentration which occurred in four of the children with perceptive deafness would suggest that vitamin K did not act, as has been suggested, by causing haemolysis and increasing the degree of hyperbilirubinaemia. Brodie and Ballantine, working on bacteria and not on animals, have suggested that synkavit may uncouple oxidative phosphorylation. This is the same activity that bilirubin is known to possess (Ernster *et al.*, 1957), and if it can be shown to do the same in animals it may explain the damage which the combination of bilirubin and vitamin K may produce.

The hearing loss from hyperbilirubinaemia evidently results from damage to the primary nuclei in the auditory pathway (the cochlear nuclei). No evidence is forthcoming of damage to the peripheral receptor, but various histological studies have shown staining of the cochlear nuclei, and the nuclei in the floor of the fourth ventricle are among the structures most likely to be affected.

There are also physiological reasons why the cochlear nuclei can be selectively damaged, as was pointed out in previous studies (Fisch and Osborn, 1954; Fisch, 1955). This is an area which, in the newborn infant, may have the highest metabolic rate of all the structures in the central nervous system. It is now thought that kernicterus is the result of metabolic damage to the central nervous system caused by bilirubin, and vitamin K may act in a similar fashion. It is not surprising, then, that an area which has a very high metabolic rate may be selectively damaged, producing an isolated clinical condition, such as perceptive deafness.

Summary and Conclusions

50 children who were jaundiced at birth, and who had regular daily estimations of total bile pigments and conjugated bilirubin, had their hearing fully tested later, and in six cases a typical perceptive partial deafness was confirmed.

The results showed that perceptive deafness of a characteristic type may occur in jaundiced babies in the absence of athetoid cerebral palsy and at a bilirubin concentration lower than generally regarded as dangerous.

The findings suggest, however, that hyperbilirubinaemia may not have been the sole factor in these cases, and that overdosage with vitamin K may have been partly responsible, although there may have existed some other, so far unsuspected, circumstances which contributed to the damage resulting in deafness.

Further studies will be necessary to find out whether the safety margin of bilirubin levels, applicable to the development of kernicterus and athetoid cerebral palsy, does in fact apply to development of perceptive deafness.

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REFERENCES

Allison, A. C. (1955). *Lancet*, **1**, 669.
Claireaux, A. E., Cole, P. G., and Lathe, G. H. (1953). Ibid., **2**, 1226.
Crabtree, N., and Gerrard, J. (1950). *J. Laryng.*, **64**, 482.
Ernster, L., Herlin, L., and Zetterström, R. (1957). *Pediatrics*, **20**, 647.
Fisch, L. (1955). *J. Laryng.*, **69**, 479.
—— (1957). *Speech*, **21**, 43.
—— and Osborn, D. A. (1954). *Arch. Dis. Childh.*, **29**, 309.
Lathe, G. H., Claireaux, A. E., and Norman, A. P. (1958). In *Recent Advances in Paediatrics*, edited by D. Gairdner, Chapt. 3, p. 87. Churchill, London.
—— and Ruthven, C. R. J. (1958). *J. clin. Path.*, **11**, 155.
Laurance, B. (1955). *Lancet*, **1**, 819.

"If we believe that the typical general hospital of the second half of the twentieth century is the hospital of 400 to 800 beds, affording all the main specialties, and that the little hospitals, often provided with such loving care a century and more ago, are to-day for the most part unfair to the patient and unattractive to staff, then we must dare to express our conviction in the hospital pattern which we plan. None of all this will be easy. We shall find old habits, traditions, prejudices, mistaken loyalties embattled across our path, and the pattern of administration and the complexities of salaries and gradings will add to our difficulties. But against all this we possess a sovereign remedy, and an invincible ally, if we will call it to our aid. It is this. The public do not want a second-rate hospital service. They want the best. They want the most enlightened and effective handling of sickness in all its forms: they want modern methods, modern buildings, modern techniques, modern medicine. Let them only be convinced that these are the standards by which we have framed our plans, and they will brush aside with impatience the opposition of prejudice or self-interest." (Minister of Health, Mr. ENOCH POWELL, addressing the Association of Hospital Management Committees, June 16.)

CHILDHOOD OBESITY

A LONG-TERM STUDY OF HEIGHT AND WEIGHT

BY

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There are only a few studies of the natural history of childhood obesity. Mullins (1958) found about one-third of adults attending his out-patient department to be overweight, and in one-third of these the obesity dated from childhood. The only long-term prospective study of childhood obesity appears to be that of Haase and Hosenfeld (1956). It was based on 335 overweight children first examined between 1936 and 1940. Fifty of these patients were re-examined after the war, when the majority were between 20 and 36 years of age. Four-fifths of the patients were still grossly overweight and only 11 were less than 20% overweight. Whereas at the first examination no patient had been more than 80% overweight, at the follow-up examination three were more than 80% overweight. The prognosis tended to be worse for the girls than for the boys.

We present the results of a nine-year prospective study of the weights and heights of a group of 98 overweight children.

Patients and Methods

In 1950 a clinic for the treatment of overweight children was started at the Children's Hospital, Birmingham. Our observations are based on the first 98 children to attend this clinic with simple obesity.

The patients were re-examined in 1951, 1956, and 1959. The findings in 1950 and 1951 have already been reported (Wolff, 1955). During this first period treatment consisted of a 1,000-calorie diet and amphetamine sulphate, and most of the children attended the clinic regularly. After 1951 only a few attended regularly. Table I gives the number of patients followed up on each occasion and the mean age and range of the group. A total of 89 of the original 98 children were re-examined in 1956 and/or 1959. Of the nine patients whom we were unable to contact in 1956 or 1959, six had already been lost sight of in 1951.

TABLE I.—*The Follow-up*

Year	Boys		Girls		Total No.
	No.	Mean Age and Range	No.	Mean Age and Range	
1950	39	9 yrs 4 mths (1 yr 4 mths–14 yrs)	59	9 yrs (1 yr 10 mths–13 yrs)	98
1951	33	10 yrs 9 mths (3–15 yrs)	49	10 yrs 3 mths (4–17 yrs)	82
1956	33	14 yrs 6 mths (7–19 yrs)	52	14 yrs 8 mths (7–20 yrs)	85
1959	26	18 yrs 2 mths (10–23 yrs)	41	17 yrs 7 mths (11–23 yrs)	67

Heights.—The height was measured with a stationary measure having a sliding headarm except when the examination took place in the patient's home, when a flexible metal tape-measure was used. The height was recorded to the nearest quarter-inch with the patient erect and barefoot.

Weights.—The weight was taken with platform scales of the spring-balance type and recorded in pounds. At the first two examinations the children were weighed without clothes except pants. At the last two examinations weights were taken in indoor clothes without jacket and shoes. Correction for the weight of the clothes was made from Widdowson's (1947) table.

Eleven of the 85 patients contacted in 1956 and 6 of the 67 contacted in 1959 were not seen by us but recorded their heights and weights themselves.

Standards Used

In the previous study (Wolff, 1955) we used the standards for weight and height of Engelbach (1932), which up to the age of 13½ are in close agreement with recent standards for English children (Daley, 1950). Engelbach's height-for-age data for children up to 13½ years also agree closely with the most recent standards for English children by Tanner (1958). But for children over the age of 13½ Engelbach's standards no longer apply to post-war English children, who are now significantly taller than the pre-war generation. In our study of the ultimate height reached by obese children we have therefore used Tanner's data as a standard. These probably represent the most reliable data available for the age-group 17–18, though Tanner himself points out that they are not based on measurements, but are arrived at by extrapolation. Tanner's data as regards boys are in close agreement with those obtained by Morant (1952) for apprentices in the Royal Air Force. To confirm the suitability for our purposes of Tanner's standard for girls we obtained the heights of 105 English nurses aged 17–19 years inclusive in training at the Birmingham Children's Hospital. Their mean height was 64.3 in. (163.3 cm.) (standard deviation: 2.33 in. (5.9 cm.)). This figure is again in fair agreement with Tanner's standard.

When studying the prognosis of the obesity, we have estimated overweight by comparing the actual weight with the standard for the patient's sex and height and have expressed the result as a percentage. In these calculations Engelbach's weight for height tables were used. The use of Engelbach's standard in this connexion is justified by the fact that, despite the well-documented secular trend for the growth spurt to occur earlier (Tanner, 1955), the relationship between weight and height has remained relatively constant over the years.

Results

Ultimate Height

Figs. 1 and 2 show the height of the patients at the first and last examinations. For clarity and convenience we have excluded the 13 children who were under 5 years at the first examination and the 7 who were under 14 at the last examination. At the first examination the patients had either not yet entered puberty or were passing through the years of puberty. It has already been shown (Wolff, 1955) that at this time the children were significantly taller than the standard. At the last examination, nine years later, most of the patients had completed puberty and had reached an age when growth in height has usually ceased (Clements, 1954). Their height was now below that expected for individuals of the same age and sex. The 25 girls who were over 17½ years of age and in whom growth in height had therefore ceased completely, or almost so, had a mean height

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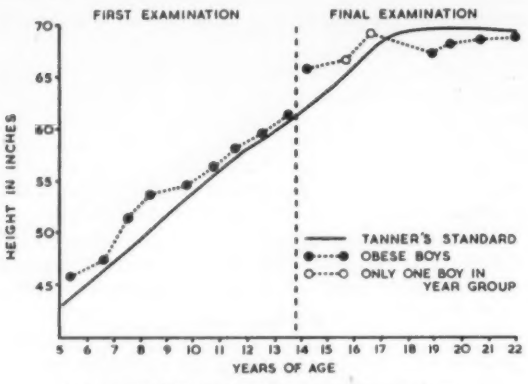

FIG. 1.—Height in relation to age for the boys.

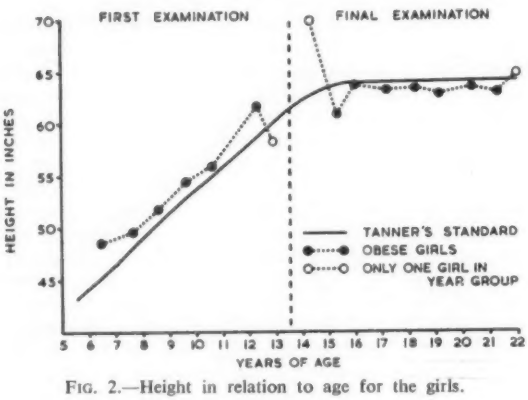

FIG. 2.—Height in relation to age for the girls.

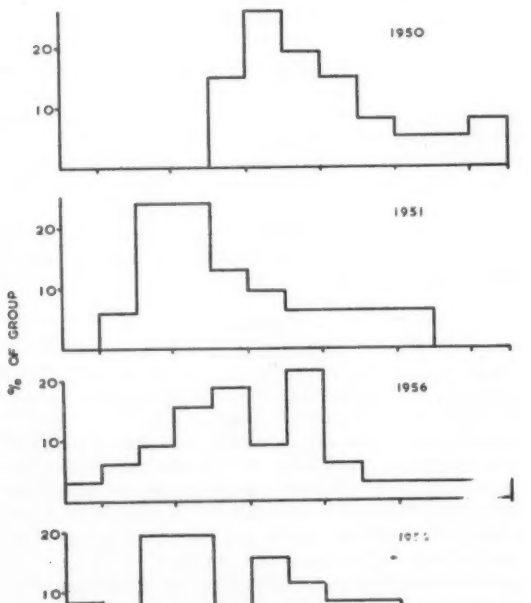

FIG. 3.—Percentage overweight for the boys.

of 63.17 in. (160.4 cm.) (standard deviation: 2.11 in. (5.36 cm.)) compared with Tanner's standard of 64 in. (162.5 cm.) ($t=1.98$, $P=0.05$). The 19 boys who were over 18 years and in whom growth had therefore ceased completely, or almost so, had a mean height of 68.16 in. (173.1 cm.) (standard deviation: 2.98 in. (7.6 cm.)) compared with Tanner's standard of 69 in. (175.2 cm.) ($t=1.23$, $P=0.2$). The mean under-height of the two sexes, taken together, is 0.835 in. (2.22 cm.) (standard deviation: 2.49 in. (6.3 cm.)), and this differs significantly from Tanner's standard ($t=2.22$, $P=0.05$–0.02).

Further analysis of our data suggests that the patients who at the first examination had been much above the expected height tended to reach an ultimate height close to the standard, whereas the patients who at the first examination had been of standard or even below standard height tended to have an ultimate height below the standard.

Prognosis of the Obesity

Figs. 3 and 4 show the percentage overweight for height in 1950, 1951, 1956, and 1959 for boys and girls respectively. Table II gives the mean overweight and standard deviation.

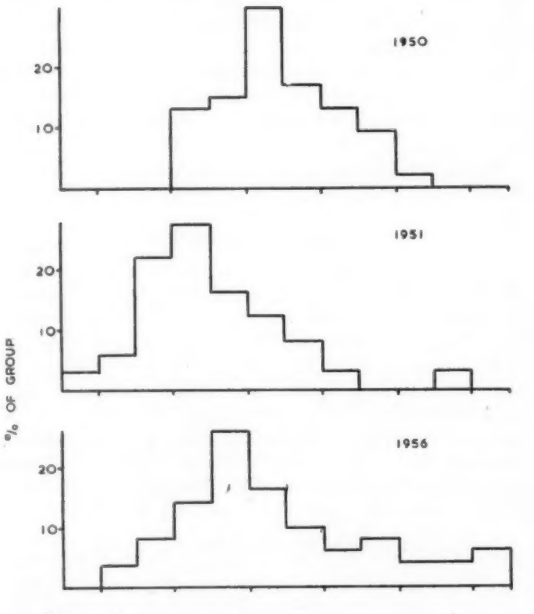

FIG. 4.—Percentage overweight for the girls.

TABLE II.—*Prognosis of Obesity*

Year	Boys		Girls	
	Mean Overweight (%)	Standard Deviation	Mean Overweight (%)	Standard Deviation
1950	63	29·8	58	15·6
1951	35	23·5	30	17·8
1956	44	27·7	50	30·4
1959	35	24·2	48	33·5

During 1950 and 1951, when the patients received intensive treatment, a highly significant reduction in mean overweight occurred in both sexes (boys : $t=4.51$, $P=0.001$; girls : $t=8.62$, $P=0.001$). However, between 1951 and 1956 a relapse occurred which was highly significant in the girls ($t=3.30$, $P=0.001$), but failed to reach statistical significance in the boys ($t=1.38$, $P=0.1$–0.2). Between 1956 and 1959 no significant change took place in either sex, though in the boys there was a tendency to improvement.

Table III gives the mean overweight at the last examination for boys over 18 and girls over 17½, when growth had presumably ceased. The group is divided into two : those over and those under 20 years of age. The older age-group was on average more overweight than the younger, and the difference between the two groups, taking the sexes together, is significant ($t=2.0$, $P=0.05$).

TABLE III.—*Overweight of Girls Over 17½ Years and Boys Over 18 Years*

		No.	Mean Overweight %	S.D.
Boys	18–19 years	13	33·5	19·87
	20 years and over ..	7	41·6	23·0
Girls	17½–19 years	14	41·0	28·65
	20 years and over ..	10	65·8	35·8

Comparison of the weights at the last examination of the patients who at the first examination had been least overweight (30–39%) with the weights of those who had been most overweight (over 80%) shows that a highly significant difference ($t=2.66$, $P=0.02$–0.01) had persisted (Table IV).

TABLE IV.—*Relationship Between Overweight at First and Last Examinations*

	Overweight at 1st Examination	
	30–39%	Over 80%
Number	11	13
Mean overweight at last examination ..	44	78
Standard deviation	24·7	37·6

Of the 13 children who were grossly overweight at the first examination, only one had returned to near normal weight. This was a girl who at the beginning of treatment was 4 years old and 98% above expected weight. Fifteen months later she was only 26% overweight, and at the final examination, when 12½ years old, she was 10% overweight. The remaining 12 were still grossly overweight (range, 47–153%). Of the 11 children who at the first examination were least overweight, only 3 were less than 20% overweight at the final examination ; the remaining 8 were more than 45% overweight.

A few findings of negative prognostic significance can be summarized : the rate at which the patients lost weight during the period of intensive therapy between the first and second examinations bore no relationship to the degree of overweight at the final examination. In fact, the nine children who during the initial period lost weight at a monthly rate of more than 4% of their expected weight were at the final examination on average 78% above expected weight, and only one of them had returned to near normal weight.

The mean overweight at the final examination of the patients whose weight after intensive treatment had returned to near normal—that is, less than 20% overweight—did not differ significantly from that of the children whose weight had responded less satisfactorily to treatment. In fact, of the 20 children who at the second examination were less than 20% overweight, only 4 were less than 20% overweight at the final examination. In other words, in four-fifths the condition had relapsed.

Age at the first examination had no bearing on the prognosis.

Discussion

The finding that obesity in childhood is likely to persist into adult life is in agreement with that of Haase and Hosenfeld (1956), and, like these authors, we find that the outlook may be a little better for boys than for girls. The influence of overweight on the morbidity and mortality of adults is well recognized, and there is reason to believe that weight reduction brings improved health and longevity (Marks, 1960). A better understanding of the natural history of obesity thus leads to a greater emphasis on the treatment of the condition in childhood. In this connexion we wish to stress the great importance, after initial weight reduction, of long-continued supervision to prevent relapse. Our experience with the grossly overweight child—that is, more than 50–60% above the expected weight—suggests that hospitalization may often be necessary initially to achieve satisfactory weight reduction.

Previously (Wolff, 1955) it was shown that obese boys and girls tend to be above average height compared with children of all social classes, but that their height does not differ significantly from children of the upper social classes. It was also shown that they enter puberty about one year earlier than average. One reason for the present study was to determine the ultimate height reached by the overweight child. As Tanner (1955) states, final size and the speed with which it is attained are not necessarily related. Studying boys, he concludes that there is a strict relationship between adult height and maturity group, the latest maturers being the tallest, and Stone and Barker (1937) found that later-maturing girls are somewhat taller than average. It is then not surprising that the ultimate height of the obese child, who is known to mature early, is shorter than average, even though until puberty he was taller.

Summary

In a nine-year prospective study of a group of 98 children with simple obesity their ultimate height was found to be significantly below standard height, even though before the onset of puberty their height had been above the standard.

There was a strong tendency for the obesity to recur after initial weight reduction and then to persist into young adult life. At the last examination only one-quarter of the group had achieved normal or near normal weight (less than 20% overweight) and among the girls the proportion of grossly overweight patients (above 80% overweight) was greater than at the first examination. Energetic treatment of obesity in childhood followed by prolonged supervision to prevent relapse is important in the prevention of obesity in the adult with its well-recognized influence on morbidity and mortality.

We are grateful to Miss M. Woods, our matron, for allowing us access to the height and weight records of the nurses.

REFERENCES

Clements, E. M. B. (1954). *Arch. Dis. Childh.*, **29**, 147.
Daley, A. (1950). L.C.C. Report on the Heights and Weights of School Pupils in the County of London in 1949. L.C.C., London.
Engelbach, W. (1932). *Endocrine Medicine*, **1**, 304. Thomas, Springfield, Ill.
Haase, K.-E., and Hosenfeld, H. (1956). *Z. Kinderheilk.*, **78**, 1.
Marks, H. H. (1960). *Bull. N.Y. Acad. Med.*, **36**, 296.
Morant, G. M. (1952). Air Ministry, Flying Personnel Research Committee, Report No. 711f (unpublished).
Mullins, A. G. (1958). *Arch. Dis. Childh.*, **33**, 307.
Stone, C. P., and Barker, R. G. (1937). *Hum. Biol.*, **9**, 1.
Tanner, J. M. (1955). *Growth at Adolescence*. Blackwell, Oxford.
—— (1958). In *Modern Trends in Paediatrics*, edited by A. Holzel and J. P. M. Tizard, p. 325. Butterworth, London.
Widdowson, E. M. (1947). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 257, p. 10.
Wolff, O. H. (1955). *Quart. J. Med.*, **24**, 109.

TUBERCULOSIS IN SOHO

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Of the 3,105 catering establishments in the City of Westminster over 400 are concentrated in Soho. Tinker (1959) suggested that workers in the catering trade in this area have a particularly high incidence of pulmonary tuberculosis. The present survey was made to try to find out the true position and to answer certain questions. For instance, is tuberculosis usually contracted before or after joining the trade ? If before, is it because catering attracts workers whom tuberculosis has incapacitated for other types of work, or is it, in the case of workers from abroad, because they already have the disease when they arrive in Britain ? If after, is it because previously uninfected workers get their primary infection after entering the crowded working conditions of the trade, or is it simply that those who have already had their primary infection break down as a result of adverse working or living conditions ?

Method

The area chosen for the inquiry was the electoral ward of Soho, which covers about 1/7 square mile (0.37 sq. km.) in the City of Westminster. The names of some 400 catering establishments were provided by the Public Health Department and 240 were included in the survey. There were 47 public-houses and 193 restaurants, cafés, and coffee-bars. It was not administratively possible to include the clubs, staff canteens, and theatre bars which made up the total figure.

A personal letter was written to each proprietor, manager, or licensee, explaining the purpose of the survey and asking him to supply the names of his staff so that each individual could be sent an explanatory letter and an appointment for an *x*-ray examination. Replies to these letters were received from 105 establishments ; 96 gave the names of their staff and 9 gave numbers only. One of us (G. B.) visited the 135 employers (56%) who did not reply, and on this approach a further 99 gave the names of their staff and 34 gave numbers, thus leaving only two establishments from which no information could be obtained. In this way the total sample was estimated to be 4,733 workers.

Arrangements were made with the South-West London Mass *X*-ray Service for one of their mobile units to make 12 visits to various sites in Soho between January and June, 1960, and for their static unit to make a four-day visit to one large establishment which employed approximately 1,800 people. It was not possible to send each of these 1,800 workers an individual letter, but the management publicized the visit and encouraged their employees to attend during working hours ; 930 (52%) of the 1,800 were interviewed and *x*-rayed.

Each of the sites chosen for the mobile unit was as near to as many restaurants as possible. About four days before each visit of the unit an explanatory letter was sent to each person in the appropriate restaurants telling them the time and place for their *x*-ray examination. In this way personal letters were sent to 2,933 workers and 1,319 (45%) kept the appointment to be interviewed and *x*-rayed. Those who failed to attend were sent another appointment and asked to state whether they were already attending a chest clinic or hospital. Information and *x*-ray films were obtained for a further 362 (12%).

Thus, of the 4,733 workers approached in various ways, 2,611 (55%) responded. Before being *x*-rayed each person was interviewed and helped to complete a questionary answering the following questions: Age ? Sex ? Address ? Occupation ? Place of work ? In which country did you spend your childhood ? How many years have you spent in the catering trade ? How long have you been at this café/restaurant/public-house ? How long have you been in Great Britain ?

Those workers whose miniature chest radiographs were normal were so informed and those with abnormal films were recalled for further radiological and clinical assessment at Westminster Hospital Chest Clinic. After any necessary investigations the workers with radiographic evidence of pulmonary tuberculosis were classified as follows:

1. Active pulmonary tuberculosis requiring treatment with antituberculous drugs: (*a*) sputum negative for tubercle bacilli, (*b*) sputum positive for tubercle bacilli.

2. Pulmonary tuberculosis not requiring active treatment but requiring observation at clinic.

3. Healed pulmonary tuberculosis not requiring observation and therefore discharged from further attendance.

In the patients who showed evidence of pulmonary tuberculosis an assessment was also made of the duration of the disease, with particular reference to the time of joining the catering trade and, in the case of immigrants, the time of arrival in Great Britain. This assessment was made on the clinical history, the radiographic appearances, and the history of previous radiographic examinations.

Results

Of the 2,611 workers examined, 86 showed radiological evidence of past or present tuberculosis. The 86 cases were divided into three groups. In the first or "active" group there were 21 patients suffering from active tuberculosis requiring treatment with antituberculous drugs: sputum examinations for tubercle bacilli were positive in 6 and negative in 15. In the second or "observed" group there were 32 patients with tuberculosis requiring clinic observation only ; two of these patients were already attending a chest clinic elsewhere.

In the third or " discharge " group there were 33 patients with evidence of old healed pulmonary tuberculosis not thought by us to require clinic observation. Even so, 11 of these were in fact attending other chest clinics.

Table I summarizes these findings and shows the incidence of these three types of tuberculosis. The most important figure is that for active pulmonary tuberculosis —8.0 per 1,000 workers *x*-rayed.

TABLE I.—*Classification of the 86 Workers who Showed Radiographic Evidence of Past or Present Pulmonary Tuberculosis*

Description of Disease	No. of Cases	Incidence of Pulmonary Tuberculosis per 1,000 Workers *X*-rayed
" Active " (all new cases) { sputum T.B.–negative	15	8·0
" " " –positive	6 } 21	
" Observed " group (including 2 already attending other chest clinics)	32	12·3
" Discharged " group (including 11 already attending other chest clinics)	33	12·6

TABLE II.—*Incidence of Pulmonary Tuberculosis per 1,000 Workers According to Occupation in the Catering Trade*

Occupation	Active Groups	Active and Observed Groups	Active, Observed, and Discharged Groups	No. *X*-rayed
Employers and managers	8·6	17·2	51·5	233
Food-servers	6·2	17·3	27·2	809
Food-preparers	10·6	25·8	39·5	659
Other kitchen staff	13·6	27·3	38·2	367
Alcohol-servers	12·7	25·3	25·3	158
Office staff	—	10·9	19·1	366
Occupation unknown	—	—	—	17
All occupations	8·0 (21 cases)	20·3 (53 cases)	32·9 (86 cases)	2,611

Table II shows the incidence of tuberculosis in the various occupational classes in the catering trade. It will be seen that active tuberculosis is particularly common in alcohol-servers, food-preparers, and other kitchen staff. The incidence in the employers and managers is also surprisingly high.

In so cosmopolitan a community as Soho it is important to assess the various national groups separately, and Table III shows the incidence of tuberculosis according to the country of origin. Of the 840 workers born in Great Britain active tuberculosis was found in four (4.8 per 1,000). Of the remaining 1,771 workers, all of whom were immigrants, active tuberculosis was found in 17 (9.6 per 1,000). Thus active tuberculosis was twice as common in the immigrants as a whole. The Chinese, who mostly come from Hong Kong, had the highest incidence of all (53.8 active cases per 1,000). They are responsible for the increased incidence of active disease in the immigrants. If the Chinese are excluded the incidence of active disease among the rest of the immigrants is only 4.4 per 1,000 workers, a lower incidence than in those born in Great Britain.

TABLE III.—*Incidence of Pulmonary Tuberculosis per 1,000 Persons According to Country of Origin*

Country of Origin	Active Group	Active and Observed Groups	Active, Observed, and Discharged Groups	No. *X*-rayed
Ireland	—	15·9	26·5	189
Italy and Malta	2·7	10·7	32·1	374
Cyprus	3·6	21·7	29·0	276
Pakistan, India, and Burma	6·2	18·6	24·8	161
China and Hong Kong	53·8	91·4	118·3	186
West Indies	—	3·7	3·7	272
Other European countries	12·6	29·4	46·2	238
Elsewhere	13·3	26·7	26·7	75
All the above countries	9·6 (17 cases)	24·3 (43 cases)	36·7 (65 cases)	1,771
Great Britain	4·8 (4 cases)	11·9 (10 cases)	25·0 (21 cases)	840
All countries	8·0 (21 cases)	20·3 (53 cases)	32·9 (86 cases)	2,611

In each of the 86 workers who showed evidence of past or present pulmonary tuberculosis an attempt was made to decide whether they had developed the disease before or after joining the catering trade. In 17 there was insufficient evidence to make such a decision ; in 14 it was certain that they had developed the disease after joining the catering trade, because they had had a normal chest radiograph at or since that time ; in 24 who had not previously been *x*-rayed there was clinical evidence that they had probably developed the disease after joining the trade. Similarly there was definite radiological evidence that 23 had the disease before joining the trade and clinical evidence that another 8 probably had it before then. Table IV shows the overall picture ; 31 workers definitely or probably developed tuberculosis before joining the catering trade ; 38 definitely or probably developed tuberculosis after joining the catering trade.

TABLE IV.—*Development of Tuberculosis Before or After Joining the Catering Trade, According to Nationality*

Country of Origin	No. of Patients who, on Entering the Catering Trade: Were Not Tuberculous (on Radiological Evidence)	Probably Were Not Tuberculous (on Clinical Evidence)	Were Tuberculous (on Radiological Evidence)	Probably Were Tuberculous (on Clinical Evidence)	No Decision Possible
Ireland	—	1	2	—	2
Italy and Malta	1	1	4	2	4
Cyprus	1	4	1	1	1
Pakistan and India	—	2	1	1	—
China and Hong Kong	4	4	7	4	3
West Indies	—	—	1	—	—
Other European countries	3	3	3	—	2
Elsewhere	—	2	—	—	—
All above countries	9	17	19	8	12
Great Britain	5	7	4	—	5
All countries	14	24	23	8	17
	38		31		

Although this distribution is fairly even, Table IV shows that marked differences appear when the figures are broken down for the various nationalities. The following ratios include the patients in whom there was radiological evidence of the presence or absence of tuberculosis at the material times, as well as those in whom the evidence was based on the clinical history only. The same order of results is obtained if only the cases with radiological evidence are considered.

Tuberculosis appeared more often after than before joining the trade in those from Great Britain (12 : 4), in those from Cyprus (5 : 2), and in those workers listed as coming from other European countries (6 : 3), and elsewhere (2 : 0).

Tuberculosis appeared more often before than after joining the trade in those from China and Hong Kong (11 : 8), in those from Italy (6 : 2), in those from Ireland (2 : 1), and in those from the West Indies (1 : 0). The disease appeared just as often before as after joining the trade in those from India and Pakistan (2 : 2).

The numbers in Table IV refer to the time of joining the catering trade, whether in this country or abroad, but, although some of the workers from abroad were in the catering trade before coming to the United Kingdom, none developed tuberculosis in the catering trade abroad. Exactly the same numbers are therefore obtained whether the information is considered with reference to either joining the catering trade in the United Kingdom or simply coming to the United Kingdom.

Table IV is based on all the cases of tuberculosis whether active, observed, or inactive. To try to find out when and why tuberculosis occurred in those workers who developed the disease after joining the trade the time intervals at which the active cases were discovered after joining the trade were studied. Table V shows this as the incidence of active tuberculosis per 1,000 workers according to the number of years in the catering trade. Because the Chinese form such a high proportion of the active as well as the inactive cases they are considered separately from the rest of the workers, the other nationals.

TABLE V.—*Incidence of Active Pulmonary Tuberculosis per 1,000 Persons According to Length of Time in Catering Trade for Chinese and All Other Nationals*

Years in Catering Trade	Chinese		Other Nationals	
	No. *X*-rayed	Incidence of Active P.T.B. per 1,000	Incidence of Active P.T.B. per 1,000	No. *X*-rayed
0 up to 2 ..	66	45	0	510
2 ,, 3 ..	25	80	22	183
3 ,, 5 ..	28	107	15	261
5 ,, 7 ..	23	87	0	210
7 or more ..	37	0	3	1,183
No reply ..	7	0	0	78
Totals ..	186	53·8 (10 cases)	4·5 (11 cases)	2,425

Only 4 of the 21 patients with active disease had been born in Britain; they had been in the catering trade for 2, 3, 34, and 35 years respectively. Their evenness of distribution in Table V means that the findings mainly reflect the incidence of active disease in the immigrants.

For those Chinese who had been in the catering trade for less than two years the incidence of active tuberculosis was 45 per 1,000 persons, whereas the incidence in the same period among the other nationals was nil.

In the subsequent years the Chinese still showed a high incidence until the beginning of the seventh year, after which there were no active cases among the few who had been in the catering trade so long. The other nationals showed an incidence of 22 and 15 active cases per 1,000 among those who had been in the catering trade between two and less than three years and between three and less than five years respectively. The incidence was only 3 per 1,000 among those who had been in the catering trade more than seven years.

Discussion

We realize that the results of this survey are based on a 55% sample which was to some extent selected. On account of the language difficulties among many different nationalities and the constant movement in the trade in Soho, even this response was obtained only by considerable effort and personal visiting. There is no reason to suppose that the 45% who did not co-operate were any less likely to have tuberculosis. In fact, there must have been many who knew that they had tuberculosis or who were already attending clinics and who deliberately avoided the survey for fear of losing their work as food-handlers.

We know of no other *x*-ray survey figures for the incidence of tuberculosis in catering workers in Great Britain. The only other similar surveys were done in the United States over 10 years ago (Martin *et al.*, 1934; Boucot and Sokoloff, 1948). The Report of the County Medical Officer of Health for London for the year 1959 (L.C.C., 1960) gives the tuberculosis notification rates for London workers in the "personal services" (among whom are the food and restaurant workers) as 2.1 per 1,000 males and 0.6 per 1,000 females. The rates for the general population of London are 1.2 per 1,000 males and 0.7 per 1,000 females. These figures indicate that, although tuberculosis is more common in catering workers generally than in the ordinary population, it is less than twice as common.

The present study not only confirms our expectation that pulmonary tuberculosis is more common in workers in the catering trade in Soho than in the general population, but also suggests that it is commoner among these workers in Soho than in those in the catering trade elsewhere. The incidence of active disease in Soho was found to be 8.0 per 1,000 workers. The most useful figure with which this finding can be compared is the incidence of active pulmonary tuberculosis found in the general public by the South-West London Mass *X*-ray Service in 1959; this was 1.8 per 1,000 persons *x*-rayed (F. A. Nash, 1959, personal communication). The definition of active tuberculosis used for such returns made by chest physicians to mass *x*-ray units is "tuberculosis requiring treatment or close clinic supervision." The phrase "close clinic supervision" has never been defined, and is interpreted differently by different physicians; the definition used in those returns is wider than the one used in this survey, and in the present context over-estimates the number of cases of active tuberculosis in the general population. Thus, comparison of this figure of 1.8 per 1,000 for the general population with the figure of 8.0 per 1,000 for the workers in the catering trade actually underestimates the increased incidence of the disease in these workers in Soho.

Active tuberculosis is therefore well over four times as common in workers in the catering trade in Soho as in the general population, and almost certainly more common than in the catering trade generally.

Some possible explanations for this high incidence were put forward in the introduction to this paper, but the results of the survey show that none of these simple explanations wholly accounts for the situation. Although the workers born in Great Britain have an incidence of active tuberculosis which is more than twice that found in the general population, the immigrants clearly account for most of the tuberculosis in the catering trade in Soho. Although the immigrants as a whole suffer from tuberculosis twice as commonly as the British-born workers, it is not just a matter of the immigrants arriving in this country with tuberculosis, because only about half the affected immigrants had the disease before joining the trade in Great Britain.

The Chinese and to a less extent the Italians and the Irish usually developed tuberculosis before joining the catering trade, and these are all countries with a high national incidence of tuberculosis according to the published mortality rates per 100,000 living persons for 1957–8. In Hong Kong the rate for that year was 103.0, in Italy 21.0, and in Ireland 24.0, compared with 8.9 in

England and Wales (Saliba, 1960). Active tuberculosis is apparently uncommon in West Indian immigrants; there was no active tuberculosis among the 272 West Indians in the present study, and only one showed inactive disease which he had before coming to Great Britain. In a mass *x*-ray study of 978 West Indians in North-West London, Brett (1958) found only two cases of active tuberculosis—an incidence no higher than that in the general population. The incidence of tuberculosis varies in the different islands of the West Indies, but in Jamaica the mortality rate was 24.0 per 100,000 in 1955 (K. Robson, 1960, personal communication), whereas the figure was 13.1 in England and Wales in the same year (Registrar-General, 1960). The disease appeared just as often before as after joining the trade in those from India and Pakistan, and in these countries the mortality rates for tuberculosis are also high (Heaf, 1957). It seems likely that most of the workers who come from any of these countries have usually already been infected before they arrive in Great Britain and join the catering trade.

The Cypriots, the British, and to a less extent the workers listed as coming from other European countries and elsewhere usually developed the disease after joining the trade. The tuberculosis mortality rates in Cyprus are low (Constantinides, 1958) and the prevalence of infection is low (Bardswell, 1939). It seems likely that many of the workers from these countries acquire their initial infection after joining the trade.

Some support for this idea comes from the incidence of active tuberculosis expressed according to the number of years spent in the catering trade in Table V. Because 10 of the 21 cases of active tuberculosis were Chinese from Hong Kong, and because Hong Kong has such a very high incidence of tuberculosis, the Chinese were considered separately from the other nationals. The fact that a high incidence of active tuberculosis was found among the Chinese who had been in the catering trade less than two years, as well as among those who had been here for longer, is strong evidence that many of those whose disease was active had their infection before entering the catering trade in this country.

In contrast there were no cases of active tuberculosis among any of the other nationals who had been in the catering trade less than two years. Their highest incidence was among those who had been in the trade between two and less than five years; this suggests that most of them arrive healthy or with inactive disease but break down within the first five years after their arrival.

Let us emphasize that we are now talking about all the workers other than the Chinese. There are two possible explanations for the described incidence of active disease in this majority group. The first is that the worker arrives already tuberculin-positive and then breaks down as a result of adverse conditions in the trade. This is possible but perhaps unlikely, because most of the workers have come from abroad to a higher standard of living in this country; it may apply to some of those workers from countries such as Italy and Ireland with a high national incidence of tuberculosis.

The second and more likely explanation is that many of the workers originating from countries with a low national incidence of tuberculosis, such as Cyprus, the United Kingdom, and other European countries, join the trade tuberculin-negative, but soon receive their primary infection in the pool of infection which undoubtedly exists in the trade.

It seems likely that if this survey were repeated the yield of tuberculosis would again be high because of the constant movement in the trade. The frequent changes in population probably ensure that the pool of infection is constantly fed by tuberculous workers coming from countries with a high national incidence of tuberculosis.

Much has been written about the advantages and difficulties of routine radiographic examination of immigrants to this country. So far as the catering trade in Soho is concerned, it would clearly be to everyone's advantage if this were carried out at least for the Chinese. As the incidence of tuberculosis is as high in those workers born in Great Britain as in the immigrants other than the Chinese, it is clearly advisable that all new employees in the catering trade in Soho should be *x*-rayed and tuberculin-tested and, where necessary, vaccinated with B.C.G. This we have already started to do for some of the Chinese restaurants.

Summary

2,611 workers in the catering trade in Soho have been *x*-rayed and at the same time interviewed and helped to complete a questionary. From the information obtained the following conclusions have been reached.

Tuberculosis is more than four times as common in the catering trade in Soho as in the general population and almost certainly commoner than in the catering trade elsewhere in Great Britain. The disease is commonest among alcohol-servers, food-preparers, and other kitchen staff. The overall incidence is 8.0 cases of active tuberculosis per 1,000 workers *x*-rayed. The disease is twice as common among the immigrants (9.6 per 1,000 *x*-rayed) as among those born in Great Britain (4.8 per 1,000 *x*-rayed). The Chinese, who come mainly from Hong Kong, have by far the highest incidence of active disease (53.8 per 1,000 *x*-rayed). They are responsible for the increased incidence in the immigrants as a whole. The incidence of active disease in immigrants, excluding the Chinese, was 4.4 per 1,000 workers, a lower incidence than in those workers born in Great Britain.

There is evidence that rather more than half the tuberculous workers have the disease before joining the trade, and the majority of these come from Hong Kong and to a less degree from Italy and Ireland, where there is a high national incidence of tuberculosis. Those tuberculous workers born in Great Britain and others from areas such as Cyprus, where the incidence of tuberculosis is low, usually join the trade healthy but develop the disease in the first five years after coming to this country and joining the trade.

It is considered that all new entrants to the catering trade in Soho should be *x*-rayed and tuberculin-tested, and where necessary B.C.G.-vaccinated, whether they come from abroad or were born in the United Kingdom. For the Chinese these precautions are especially necessary.

We are grateful to the Dowager Countess Eleanor Peel Trust for a grant towards the expenses of the survey. We are also grateful to the Metropolitan Police of Savile Row Police Station, without whose help in obtaining parking facilities for the Mobile Units this investigation would have been impossible; to Miss Farrow, the organizing secretary, and the other staff of the South-West London Mass *X*-ray Service, and in particular the staffs of units 8C and 8K who co-operated in the study; to the Public Health Department of the City of Westminster for their invaluable assistance;

and to Miss Roy, Westminster Hospital Chest Clinic, for secretarial assistance and her help in interviewing many of the examinees.

REFERENCES

Bardswell, N. D. (1939). *Tubercle (Lond.)*, **20**, 165.
Boucot, K. R., and Sokoloff, M. J. (1948). *Amer. Rev. Tuberc.*, **58**, 684.
Brett, G. Z. (1958). *Tubercle (Lond.)*, **39**, 24.
Constantinides, M. G. (1958). Ibid., **39**, 59.
Heaf, F. R. G. (1957). *Symposium of Tuberculosis*, p. 615. Cassell, London.
London County Council (1960). *Report of the County Medical Officer of Health for the Year 1959*, p. 29. London.
Martin, D. C., Pessar, H. T., and Goldberg, J. A. (1934). *Amer. Rev. Tuberc.*, **29**, 182.
Registrar-General (1960). *Statistical Review of England and Wales for the Year 1959*, Part I, p. 43. H.M.S.O., London.
Saliba, A. (1960). *Brit. J. Dis. Chest*, **54**, 22.
Tinker, C. M. (1959). *Chest Heart Bull.*, **22**, 26.

ACUTE NEPHRITIS ASSOCIATED WITH CONGENITAL SYPHILIS

BY

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[WITH SPECIAL PLATE]

Two clinical forms of renal disease have been described in association with congenital syphilis and early acquired syphilis; these are the nephrotic syndrome and a rare form of acute nephritis, but the latter has been poorly documented. Only two cases of acute glomerulonephritis associated with congenital syphilis are recorded in the English literature (Mitchell, 1951; Yampolsky and Mullins, 1945). In view of the rarity of the condition we describe two further cases of acute nephritis in association with congenital syphilis seen in the paediatric wards of the Baragwanath Hospital.

Case 1

A well-nourished 6-weeks-old male African infant was admitted with a two-day history of blood-stained urine. Before the onset of haematuria the baby was perfectly well, and there was no history of snuffles or skin rashes. On examination he was found to be pale and extremely irritable, with marked generalized oedema. There was an icteric tinge to the conjunctivae. The heart and chest were normal, and the systolic blood-pressure was 100 mm. Hg. by the flush method. The liver and spleen were each enlarged to 2 cm. below the costal margin in the mid-clavicular line, and bilateral regular kidney masses were palpated. Examination of the urine disclosed albuminuria ++++ and innumerable red blood cells and granular casts; an excess of bilirubin and urobilin was also present.

An intravenous pyelogram showed that the pelves of the kidneys failed to fill. Radiography of the femora revealed marked periostitis Haemoglobin level 10.7 g. per 100 ml.; white cell count 52,000 per c.mm. (neutrophils 53%, lymphocytes 23%, monocytes 24%); prothrombin index 45%. Serum bilirubin 5.7 mg. per 100 ml., of which 4.1 mg. direct; blood urea 95 mg. per 100 ml., serum cholesterol 260 mg. per 100 ml. V.D.R.L. tests (Harris *et al.*, 1948) on both mother and child positive.

Treatment was begun with 250,000 units of crystalline penicillin six-hourly. During the next six days massive haematuria and albuminuria continued (results with Esbach's reagent varied between 1 g. and 5 g.), but the blood urea level fell to 54 mg. per 100 ml. On the sixth day the urinary abnormalities began to resolve; the liver had decreased in size and the spleen was no longer palpable. However, the patient became stuporous on the following day, and lumbar puncture revealed a purulent cerebrospinal fluid from which *Escherichia coli* was isolated. Despite vigorous antibiotic treatment he deteriorated steadily and died on the tenth day.

Post-mortem Findings

The body was oedematous, the oedema being most marked over the lower extremities. There was generalized pallor of the skin, and the hair was sparse and lighter in colour than normal. The heart showed moderate generalized enlargement and weighed 45 g. The kidneys were both markedly enlarged (right kidney 50 g.; left kidney 60 g.). The capsules stripped easily, leaving smooth but rather pale surfaces which had some retention of foetal lobulation. The cut surfaces of both kidneys showed marked pallor of cortex and medulla. Histological examination of the brain (520 g.) showed the presence of an acute suppurative leptomeningitis. Microscopical examination of the kidneys revealed marked changes in the glomeruli. These were swollen and showed endothelial and epithelial proliferation and infiltration by occasional neutrophils. Many glomeruli completely filled the glomerular space, and in some there were adhesions between tuft and Bowman's capsule. In addition there was slight focal thickening of the basement membrane. An occasional glomerulus showed focal eosinophilic necrosis (Special Plate, Figs. 1–3). Many tubules contained hyaline and granular casts and there were fine fat droplets in their lining cells. The blood-vessels were normal. The interstitial tissue showed an occasional focus of lymphocytic infiltration and extramedullary haemopoiesis. The histological features were those of an acute proliferative glomerulonephritis. Section of the femur failed to reveal any evidence of periostitis or epiphysitis. Spirochaetes were not isolated from any of the organs.

Case 2

An African female aged 2 months was admitted to the hospital with a history of swelling of the face and feet for one week. There was no history of haematuria, breathlessness, or cough. The mother's pregnancy had been uneventful, and delivery was at full term and normal. There had been no neonatal jaundice or cyanosis; the baby, who weighed 10½ lb. (4.65 kg.), was breast-fed.

Examination showed moderate oedema of the legs and puffiness about the eyes. Mucous membranes were moderately pale and there was no clinical jaundice. Mouth and pharynx were healthy and the heart and lungs normal. The abdomen was slightly distended, but no free fluid was present. The liver edge was palpable 2 cm. below the costal margin in the mid-clavicular line, and the spleen 1 cm. below the costal margin.

There was macroscopic haematuria and the urine contained a marked excess of albumin. Microscopical examination of the urine showed numerous erythrocytes and hyaline and granular casts. The haemoglobin level was 8.6 g. per 100 ml., and the white cell count 10,100 per c.mm. with a normal differential count. The mother gave a positive reaction to the V.D.R.L. test, but the baby's reaction was negative. Radiography of the baby's knees showed a typical syphilitic metaphysitis and periostitis. Further investigations showed: blood urea 26 mg. and serum cholesterol 245 mg. per 100 ml.; total serum proteins 5.5 g. per 100 ml., with albumin 1.3 g., α1-globulin 0.39 g., α2-globulin 0.85 g., β-globulin 0.92 g., and γ-globulin 2.04 g.; serum bilirubin 0.12 g. per 100 ml.; alkaline phosphatase 29.2 units per 100 ml.; thymol turbidity 10 units; zinc sulphate turbidity 3.2 units; thymol flocculation 2 units; zinc sulphate flocculation negative.

The patient received 5 million units of penicillin over a period of 12 days, after which the spleen and liver were much smaller but the urine still showed numerous erythrocytes and mild albuminuria. The blood urea level was then 18 mg. per 100 ml. A further 1½ million units of penicillin was given over five days, by which time the urine was free of erythrocytes, casts, and albumin and the spleen and liver were no longer palpable.

Radiography of the knees repeated 25 days after starting treatment showed no metaphysitis but still a well-marked periostitis.

Discussion

Many authors mention the association of glomerulonephritis with neonatal syphilis, but there are remarkably few fully documented reports. Standard textbooks of paediatrics make very brief mention of this association. Ruben (1959), writing on the nephrotic syndrome in children, states that "the aetiology is unknown except for relatively rare instances associated with specific diseases such as syphilis." He does not indicate with which phase of syphilis it is associated, and does not mention at all an association between syphilis and acute nephritis. Ellis (1956) makes no mention of renal involvement in neonatal congenital syphilis. He states that in juvenile congenital syphilis renal damage manifests itself by an albuminuria and occasionally by a more acute syphilitic nephritis. Sheldon (1951) holds the view that the kidneys may be involved in infantile congenital syphilis and that this "is indicated by the occasional presence of albumin, casts, and blood cells in the urine and sometimes a generalized oedema." He also considers that the kidneys may be involved in late congenital syphilis, manifesting with albuminuria, passage of casts, and occasional attacks of haematuria. Paterson and Moncrieff (1947) state that acute nephritis is rare in infancy, "but that, when it does occur, syphilis is a prominent aetiological factor because syphilitic infants are readily affected by streptococcal infections of the nasopharynx and skin." This is also the view of Rich (1932), who regards congenital syphilitic nephritis as ordinary glomerulonephritis occurring in infants who are rendered susceptible to streptococcal invasion by the congenital syphilis. Baker (1939) supports this and claims that the improvement that sometimes results from antisyphilitic treatment in these cases is due to healing of nasopharyngeal ulceration and the clearing of secondary streptococcal infection. Unfortunately antistreptolysin titres were not determined in either of our cases.

Renal disease has been shown to be not uncommon in early syphilis in adults, and Thomas and Schur (1946), reviewing 112 cases of early acquired syphilis, found 12 with evidence of renal disease, two of which had acute haemorrhagic nephritis and the remaining 10 manifested with a nephrotic syndrome. In their review Furman *et al.* (1951) confirm this pattern of the incidence of nephropathy in early acquired syphilis.

The two cases here reported showed features of both acute nephritis and nephrosis. Case 1 had features of acute nephritis, including hypertension, haematuria, and azotaemia. However, the severe albuminuria with low serum albumin and raised serum cholesterol levels in this case are usually associated with nephrosis. Case 2 had more of the features of nephrosis, but macroscopic haematuria again indicated a mixed picture. The raised γ-globulin level in this case is not seen in the usual nephrotic syndrome except when it is associated with an autoimmune disease such as systemic lupus erythematosus. This patient responded very well to antisyphilitic therapy in terms of the renal, skeletal, and reticulo-endothelial lesions. There was no evidence of nasal or pharyngeal ulceration or secondary infection in any site in either case.

There are few records of the pathological changes of congenital syphilis in the kidney. Yampolsky and Mullins (1945) describe a case of congenital syphilis showing the histological picture of acute proliferative glomerulonephritis. The appearances observed in our first case resemble their description. Potter (1952) states that a mild increase in connective tissue in the kidney is the only characteristic change of congenital syphilis, though hyalinization of glomeruli and degeneration of tubules may occur. According to Allen (1952) the kidney of congenital syphilis is characterized by interstitial inflammation in association with great numbers of treponemata in the interstitium, in the tubular lumina, and among the tubular epithelial cells.

Summary

The rare association of congenital syphilis and renal disease, particularly acute nephritis, is discussed. Two cases of infants affected with syphilitic acute nephritis are described.

We wish to thank Dr. E. Kahn and Dr. S. Wayburne for help and access to case records; the Superintendent of Baragwanath Hospital for permission to publish; the Director of the South African Institute for Medical Research for facilities granted; and Mr. M. Ulrich for the photographs.

References

Allen, A. C. (1952). *The Kidney: Medical and Surgical Diseases*. Churchill, London.
Baker, B. M., jun. (1939). *Bull. Johns Hopk. Hosp.*, **65**, 196.
Ellis, R. W. B. (1956). *Diseases in Infancy and Childhood*, 2nd ed. Livingstone, Edinburgh.
Furman, R. H., Gale, R. G., Ory, E. M., and Weinstein, A. (1951). *Ann. intern. Med.*, **35**, 444.
Harris, A., Rosenberg, A. A., and Delvecchio, E. R. (1948). *J. vener. Dis. Inform.*, **29**, 313.
Mitchell, J. (1951). *Brit. med. J.*, **1**, 396.
Paterson, D., and Moncrieff, A. (1947). *Diseases of Children*, 4th ed. Arnold, London.
Potter, E. L. (1952). *Pathology of the Fetus and the Newborn*. Year Book Publishers, Chicago.
Rich, A. R. (1932). *Bull. Johns Hopk. Hosp.*, **50**, 357.
Rubin, M. I. (1959). In *Textbook of Pediatrics*, 7th ed., ed. W. E. Nelson. Saunders, Philadelphia.
Sheldon, W. P. H. (1951). *Diseases of Infancy and Childhood*, 6th ed. Churchill, London.
Thomas, E. W., and Schur, M. (1946). *Arch. intern. Med.*, **78**, 679.
Yampolsky, J., and Mullins, D. F., jun. (1945). *Amer. J. Dis. Child.*, **69**, 163.

Important technical advances have been made in recent years in the fight against rats and mice, and the latest addition to the Ministry of Agriculture, Fisheries and Food's Bulletin series, *Control of Rats and Mice*, by R. A. DAVIS, M.Sc., D.I.C., of the Ministry's Infestation Control Division, has been written in the light of recent research. In this bulletin the author describes some of the dangers, both to health and to property, of infestation by these rodents, and various methods of planning and carrying out control operations. Some of the methods of control described are poisoning—both acute and chronic—trapping, and fumigating and gassing in holes. Detailed descriptions are given of baits, bait containers, and depositors. There are also some suggestions for proofing buildings and maintaining hygienic conditions as methods of control. (H.M.S.O., Bulletin No. 181, price 4s.; by post 4s. 4d.)

Preliminary Communications

Spontaneous Biliary Peritonitis

The aetiology of spontaneous biliary peritonitis has always remained obscure. Clairmont and von Haberer (1911) published the first comprehensive description of this condition, and were at a loss to give a reason for its occurrence. In 1913 they postulated that there must be a pathological condition of the walls of the gall-bladder or bile channels to permit an abnormal permeability. They failed to reach any conclusion, however, on exactly what this abnormality was. Sick and Fränkel (1913), Wangensteen (1926), and Burkitt (1946) described further series of cases, but none of these authors were able to give a definite opinion on the cause of the condition. Burkitt mentioned two cases described by Cope (1925–6), in which retroperitoneal extravasation of bile was a characteristic feature.

On summing up the previous literature, individual opinion appears to be divided between two possible causes: (*a*) a minute perforation of the biliary system which cannot be detected, and which has never been found at operation; and (*b*) an abnormal permeability of the biliary system for which no explanation has been offered. Both of these possibilities seem to me to be most unsatisfactory.

CASE REPORT

A woman aged 30 was admitted to hospital on April 18, 1961, with obvious signs of generalized peritonitis. Laparotomy was performed shortly after admission, and on opening the peritoneal cavity a copious quantity (about 2 litres) of bile-stained fluid poured forth. No abnormality was found in the lower abdomen. In the upper abdomen the gall-bladder was thick-walled and obviously the site of chronic cholecystitis. It was obvious by now that the bile was flowing from the lesser sac through the foramen of Winslow.

A diagnosis of spontaneous biliary peritonitis was made, and the biliary tree was thoroughly searched for evidence of a minute perforation. This included examination of the lower end of the common bile-duct by mobilization of the duodenum. No such perforation was found, but an extravasation of greenish fluid was seen around the common bile-duct and behind the duodenum on the posterior aspect of the pancreas. In this latter situation two or three small spots of fat necrosis were detected.

The lesser sac was next inspected by opening the gastro-colic omentum. It was found to contain a large quantity of bile, and the tissues overlying the pancreas were oedematous from extravasated fluid. A few more tiny spots of fat necrosis were detected. The pancreas itself was perhaps slightly larger and more oedematous than normal. The common bile-duct was opened and drained, and was found to be free from stone or other obstruction. The gall-bladder was then removed, and as I passed a cigarette drain through the foramen of Winslow into the lesser sac I noticed two or three more tiny areas of fat necrosis in the peritoneum of this area. The following tests were then carried out: serum amylase, 2,000 Somogyi units per 100 ml.; serum lipase, 1.5 units; urinary diastase, 133 units.

The results of these tests established beyond any possible doubt that our suspected case of spontaneous biliary peritonitis was none other than that of an acute oedematous pancreatitis.

DISCUSSION

Regurgitation of bile from the common duct into the pancreatic duct affords the neatest and in some ways the most acceptable explanation of the mechanism of production of acute pancreatitis (Aird, 1957). The bile acts on the pancreatic ferments, liberating serum lipase and converting trypsinogen into trypsin, thus setting in action the chain reaction of acute pancreatic disease.

No one, however, seems to have given much thought to the effect that pancreatic ferments might in turn have on the bile which has entered the pancreatic ducts. Bile consists essentially of bile salts and bile pigments bound to a complex protein molecule and thereby held in colloidal suspension, thus preventing their permeation through the walls of the biliary system.

Harkins *et al.* (1936) mentioned an important series of experiments carried out by Blad (1917), which in my opinion have a most essential bearing on the entire matter. He has shown by experiment *in vitro* that pigments in colloidal bile cannot pass through a membrane, but that when a tryptic enzyme is added the colloids are destroyed and the pigment is "liberated," and then passes through the membrane.

This, therefore, appears to me to be the essence of the entire matter: in severe cases of pancreatitis the gland is so far destroyed that any biliary exudate is obscured by haemorrhage, but in less severe cases of pancreatitis, when the reaction is less, only a small amount of pancreatic ferment is liberated: this accounts for the very few or perhaps the absence of patches of fat necrosis, and those few patches which are present may be so small and discrete as to escape detection unless carefully searched for. On the other hand, the bile is broken down by the pancreatic ferments, allowing the biliary pigments to be liberated and poured into the retroperitoneal tissues and through the peritoneum into the peritoneal cavity. On this occasion, the haemorrhagic stage not having been reached, the colour of the bile is not overshadowed by that of blood. This outpouring of bile would, of course, primarily occur into the lesser sac and thence through the foramen of Winslow into the general peritoneal cavity.

It would appear to me, therefore, that the real explanation of spontaneous biliary peritonitis is none other than that of an early or subacute oedematous pancreatitis before the haemorrhagic phase has been reached and that the amount of serum lipase liberated is so little that there may be no patches of fat necrosis or, if any are present, they are tiny and can be discovered only on the closest scrutiny.

I suggest that my explanation of this condition is the first scientifically acceptable one that has been offered, and that it may well be the correct solution of the problem.

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REFERENCES

Aird, I. (1957). *Companion in Surgical Studies*, 2nd ed., p. 1012. Livingstone, Edinburgh and London.
Blad, A. (1917). *Arch. klin. Chir.*, **109**, 101.
Burkitt, D. P. (1946). *Brit. med. J.*, **2**, 155.
Clairmont, P., and von Haberer, H. (1911). *Mitt. Grenzgeb. Med. Chir.*, **22**, 154.
—— —— (1913). *Wien. klin. Wschr.*, **26**, 891.
Cope, Z. (1925–6). *Brit. J. Surg.*, **13**, 120.
Harkins, H. N., Harmon, P. H., and Hudson, J. (1936). *Arch. Surg. (Chicago)*, **33**, 576.
Sick, C., and Fränkel, E. (1913). *Beitr. klin. Chir.*, **85**, 687.
Wangensteen, O. H. (1926). *Ann. Surg.*, **84**, 691.

A patients' school has recently been built at Normansfield, Kingston Road, Teddington, a hospital for the mentally subnormal, by the League of Friends of Normansfield. The official opening ceremony was performed by Lady BRAIN, president of the League of Friends, on June 3.

Medical Memoranda

A Case of Reiter's Syndrome in Childhood

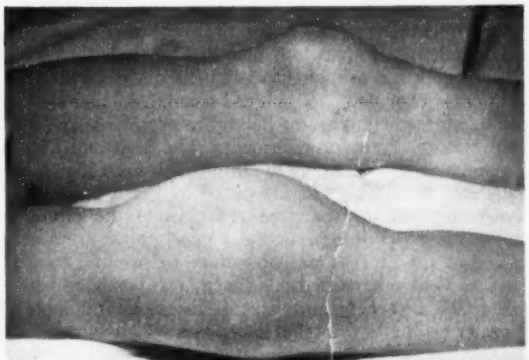
Though often reported in adults, the triad of arthritis, conjunctivitis, and urethritis has rarely been noted in children. Post-dysenteric joint manifestations have long been known, but it is only recently that cases have been described having the urethritis and conjunctivitis in association with arthritis. The first published account of this syndrome in childhood did not appear until 1946, when Koster and Jansen reported a Dutch family in which three infants developed the triad after an episode of diarrhoea. Zewi (1947) recorded a series of 10 children with the syndrome, and since then occasional cases have been reported from the United States, Finland, and Britain.

CASE REPORT

A boy aged 9 years attended the casualty department in February, 1960, complaining of pain on micturition. One week previously he had had abdominal pain associated with passage of slimy, light-coloured, loose stools, which contained no obvious blood. After passing eight motions in 48 hours both pain and diarrhoea abated without treatment. His past history was of balanitis in 1958 and acute nephritis in June, 1959; follow-up of the latter had not shown any residual renal damage. On examination his temperature was 99° F. (37.2° C.); there were no abnormal signs in the abdomen, and his penis appeared normal. His urine contained a light cloud of protein, innumerable pus cells, scanty epithelial cells, and eight red cells per high-power field. Treatment with sulphamethizole ("urolucosil") and a potassium-citrate mixture was begun and prophylactic penicillin, prescribed after his acute nephritis, was continued. He was allowed home.

Next day his temperature was 100° F. (37.8° C.). Dysuria had persisted and his right knee had become painful, possibly from an injury sustained the previous day. No swelling was noted, but the joint was most tender just below its medial margin. The urethral meatus was reddened though no discharge was seen. Treatment was not materially altered and he was sent home.

By the following day the dysuria had diminished, but the right knee was more painful and both eyes had started to discharge. He was admitted with a temperature of 101° F. (38.3° C.), oedema of both eyelids, marked conjunctivitis, and circumcorneal injection; the right knee was swollen (see photograph), with tenderness maximal on the medial surface and absolute limitation of movement. There was no urethral discharge, and the genitalia were normal, as were the cardiovascular, respiratory, and alimentary systems. He was given intramuscular penicillin 500,000 units six-hourly, and sulphacetamide eye-drops, the sulphamethizole and potassium citrate mixture being continued.



Photograph showing swelling of right knee.

Investigations.—Hb, 84%; sedimentation rate, 56 mm./hour; white blood count, 18,400/c.mm. (2% stab cells, 78% polymorphs, 14% lymphocytes, 6% monocytes). Conjunctival swab: no pathogens grown. Wassermann reaction, Reiter's protein antigen, P.P.R., and gonococcal complement-fixation test were all negative. *Brucella abortus, Salmonella typhi* O and H, *Salm. paratyphi A* O and H, *B* O and H, *C O*. Non-specific salmonella—no agglutination. Polyvalent *Shigella flexneri* serum—no agglutination. Culture of stool on monkey kidney failed to grow any cytopathogenic agent. *X*-ray examination of the knee showed a marked effusion into the joint without any bony changes. A chest *x*-ray film showed no significant pulmonary lesion. A Mantoux test was not performed because he had had B.C.G. just five weeks previously.

Within two days he recovered 5 degrees of knee movement, the temperature started to fall, and conjunctival injection decreased. A few erythematous plaques appeared on his palate the next day, and next evening a brisk epistaxis occurred controlled by adrenaline plugs. All chemotherapy was then discontinued. Now the sedimentation rate was 62 mm./hour; white-cell count, 16,600/c.mm. (63% polymorphs, 32% lymphocytes, 5% monocytes); platelets, 180,000. Knee flexion increased to 30 degrees by the tenth day, returning to normal over the subsequent three days. At the time of writing he was afebrile but complained of left palmar pain and inability to clench his fist. These signs resolved. The conjunctival injection recurred in the left eye on the twenty-first day, and in the right eye on the twenty-third day. Hydrocortisone eye-drops produced resolution within 48 hours. The sedimentation rate was then 47 mm./hour, falling in four weeks to 32 mm. and finally to 7 mm. After six weeks in hospital the patient's right knee movements were full and pain-free, and his conjunctivae were normal.

COMMENT

This condition, with which the name of Reiter has come to be associated, was originally thought to be venereal in origin, but some cases have followed a dysenteric infection. Paronen (1948), in his Finnish series, found evidence of previous *Shigella flexneri* V infection in 94% of his patients, and the case reported by Corner (1950) similarly showed a high titre of antibodies to *Shigella flexneri* V. In the present case, however, no antibodies to the *Shigella* group could be detected, and virus cultures showed no growth. The aetiology of the disorder remains obscure, but this case suggests that not all are associated with flexner dysentery. It has been suggested that Reiter's syndrome results from infection by a specific organism, as yet unidentified, gaining entry to the body as the result of another primary infection, usually dysenteric. It is also possible that the syndrome represents the reaction of susceptible individuals to certain infections, of which flexner dysentery is the commonest.

I thank Dr. R. J. K. Brown for his help and encouragement in writing this report.

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REFERENCES

Corner, B. D. (1950). *Arch. Dis. Childh.*, **25**, 398.
Koster, M. S., and Jansen, M. T. (1946). *Ned. T. Geneesk.*, **90**, 483.
Paronen, I. (1948). *Acta med. scand.*, **131**, Suppl. 212.
Zewi, M. (1947). *Acta Ophthal. (Kbh.)*, **25**, 47.

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Reviews

BEHAVIOUR THERAPY AND ABNORMAL PSYCHOLOGY

Behaviour Therapy and the Neuroses. Readings in Modern Methods of Treatment Derived from Learning Theory. Edited by H. J. Eysenck, Ph.D. (Pp. 479+xi; illustrated. 63s.) Oxford, London, New York, Paris: Symposium Publications Division, Pergamon Press. 1960.

Handbook of Abnormal Psychology. An Experimental Approach. Edited by H. J. Eysenck, Ph.D. (Pp. 816+xvi; illustrated. £6.) London: Pitman Medical Publishing Company Ltd. 1960.

Neurotic symptoms, according to Eysenck, are learned patterns of behaviour which for some reason or other are unadaptive. There is no underlying disorder. Treatment aims at the extinction of the unadaptive conditioned responses. The historical development of those habits is largely irrelevant. In Eysenck's opinion the therapist–patient relationship plays no part in the treatment, which consists in unlearning and deconditioning. The case of a child who stopped eating after her father's death is quoted as an illustration. By adopting the father's role the therapist succeeded in making the child eat again. "The theoretical rationale was that the father had become a conditioned stimulus on which eating depended." What the child felt and what the self-imposed starvation meant did not matter. A theory of neurotic behaviour which ignores so much can hardly be accepted.

Eysenck is scornful of psycho-analysts and psychotherapists who are not satisfied with "curing symptoms" but aim at treating underlying causes. But this is the aim of all medical treatment. It is nevertheless quite untrue that the removal of symptoms "is relatively unimportant to psychotherapists." All of them, including the psycho-analysts, are, in fact, only too pleased with symptomatic results, though they are not as confident of their finality as the editor of this book. The "behaviour therapist" also sees relapses, but they are regarded as new symptoms, unless they are identical with those previously observed. Thus he is able to achieve repeated final cures in the same patient, as he is concerned only with symptoms and not with people. There is no evidence that Professor Eysenck has ever carried out a treatment himself. His opinion about the irrelevance of the history of the symptoms and of the patient–therapist relationship is not shared by all contributors to this volume. Clinical observations and theories originating from psycho-analysis are dismissed wholesale. Most of the clinical contributions of his associates are concerned with apparently monosymptomatic conditions such as tics, stammer, bed-wetting, and writer's cramp and very little with the common neuroses. There is an article on deconditioning in alcoholism and a contribution by Wolpe on reciprocal inhibition as the main basis of psychotherapy. Eysenck admits that there is as yet no evidence that behaviour therapy is superior to other treatments.

The *Handbook of Abnormal Psychology* consists of 17 reviews written by the editor and his associates. In addition, there are two highly informative articles by medical authors, one on heredity by J. Shields and E. Slater, and the other on constitutional factors by L. Rees. Most of the material presented in this volume has been published elsewhere. Much of it is highly technical. The work should be useful as a reference book. Although the editor emphasizes the need for the psychologist to steer clear of psychiatric concepts, he himself and his collaborators frequently use these concepts without due definition, as if they meant the same to every psychiatrist and psychologist. Statistical methods of great sophistication are used, but often it is not clear what the authors are being precise about. It is not good enough to talk about schizophrenia and hysteria without giving operational definitions. If this is not practicable, these concepts ought not to be used by psychologists in their "independent" research.

The editor has some quaint notions about psychiatry. He divides it into two different parts, the "medical" dealing with physical diseases, and the "behavioural," which includes the neurotic symptoms. These two fields "have no underlying connexions with each other, and are as little related as surgery and epidemiology, or otology and rhinoplasty." "Medical" disorders, according to Eysenck, have a single "cause," which is absent in the behavioural disorders. It is surprising that the editor should be so much out of touch with modern medical and especially psychiatric thinking. He would be well advised to discuss these problems with his medical colleagues if a revised edition of this *Handbook* were to be contemplated. His views, presented with all the authority and force at his command, could be taken by psychologists to imply that doctors should confine themselves to the "medical" part alone and leave the "behavioural" part to the behavioural psychologists. To prevent such misunderstandings, it has to be pointed out that the division proposed by Professor Eysenck does not exist in nature. Fortunately, most of the psychologists present their contributions to this volume modestly, without trying to sit in judgment over psychiatry. They even admit that they themselves, and psychology in general, have much to be modest about. The same applies, of course, to everybody else working in the field of mental science.

E. STENGEL.

CLINICAL HAEMATOLOGY FOR STUDENTS

A Short Textbook of Haematology. By R. B. Thompson, M.D., F.R.C.P. (Pp. 306; illustrated. 30s.) London: Pitman Medical Publishing Company Limited. 1961.

The object of this book is to provide a short but readable account of clinical haematology for students of medicine. It is not intended to compete with the full accounts of large textbooks but is intended to give more information than can be found in the smaller ones. The emphasis throughout is on the clinical features of blood diseases. Laboratory techniques are not discussed at all, and morphology is for the most part left to the atlases, though four colour-plates are included. Within these limits the author has attained his objects. The accounts provided are indeed readable, manage to include a lot of important factual information, and give sensible directions for treatment. The facts and figures given are well chosen to impress the reader; for instance, he tells us that 210×10^9 red cells are needed every day just to keep the red cells in the blood at normal level. Some particularly good points are a very practical list of reasons for failure to respond to iron therapy, a chapter on anaemia associated with metabolic disorders and general diseases, and a good chapter on the haemorrhagic diseases which successfully avoids confusing the uninitiated reader with a multiplicity of "factors." Dr. Thompson adopts Dameshek's

conception of myeloproliferative disorders, though this involves including chronic myeloid leukaemia in this chapter and leaving chronic lymphatic leukaemia in a chapter on its own.

The colour plates are from paintings, but are only fair; the reproduction has too often failed to make clear the important and characteristic nuclear details. There are two radiographs and some diagrams mostly showing charts of response to treatment.

This book can be confidently recommended to the undergraduate student of medicine, and, indeed, is one of the best available accounts for this class of reader. It is sure to be put on the students' shelf in every medical library, and is sufficiently inexpensive for some students to buy their own copies.

M. C. G. ISRAËLS.

ADRENAL CIRCULATION

The Adrenal Circulation. By R. G. Harrison, M.A., D.M., and M. J. Hoey, B.Sc. (Pp. 77+vii; illustrated. 25s.) Oxford: Blackwell Scientific Publications Ltd. 1960.

This small monograph gives an account of the studies of Professor Harrison and his colleagues on the blood supply of the adrenals. The rat has been the chief experimental animal, and in addition to gross dissection of the blood vessels the technique of microradiography has been largely employed.

The main thesis of the monograph is that changes in the flow of blood through the cortex can be brought about not only by A.C.T.H. but also by constriction of the medullary arteries by humoral agents or after sympathetic stimulation. A.C.T.H. may act by causing shrinkage of cells in the zona fasciculata, with secondary dilatation of the capillaries which they adjoin. Constriction of the arteriae medullae causes blood to be diverted to the cortical vessels, and this may possibly influence cortical secretion quite independently of the hypothalamus and the anterior lobe of the pituitary. The two mechanisms of control are not necessarily antagonistic but may act synergically.

The book is well produced and contains a number of interesting observations and speculations, including the suggestion that the coming together of the originally separate cortical and medullary tissue may have been a terrestrial adaptation to meet the increased stresses of life on dry land. Presumably the initial rapid stress response results in the outpouring of adrenaline and related substances. One of the effects of these is then to shut down the blood supply to the medulla and direct the adrenal blood to the cortex, thus facilitating the cortical response. This is an attractive hypothesis, though difficult either to prove or to disprove.

J. M. YOFFEY.

MONKEYS AS EXPERIMENTAL ANIMALS

Theoretical and Practical Problems of Medicine and Biology in Experiments on Monkeys. Edited by I. A. Utkin. Translated from the Russian by Ruth Schachter, M.Sc. (Pp. 276+vii; illustrated. 63s.) Oxford, London, New York, Paris: Pergamon Press. 1960.

This book consists of a collection of papers and reports on the monkey as an experimental animal, compiled by the staff of the Institute for Experimental Pathology and Therapeutics of the Academy of Medical Science of the U.S.S.R. at Sukhumi as well as by other authorities in the Soviet Union. This medico-biological station has now been in existence for a quarter of a century with unrivalled opportunities for animal experimentation. The book contains 270 pages and is divided into three sections. The first, the largest, consists of eight reports on the physiological behaviour of several species of monkey, the majority being concerned with the central nervous system. The second part deals with spontaneous disease of monkeys, tumours, atherosclerosis, and lymphogranulomatosis and diabetes mellitus; no mention is made of infectious disease in this section. In the third section a number of experimental studies are reported—the study of measles, poliomyelitis, dysentery, and the use of antibiotics.

The subject-matter reported will be of value to readers engaged on similar lines of research, but it is strictly limited. It is unfortunate that a book such as this contains no reference to naturally occurring infections of monkeys, some of them transmissible and pathogenic to man, and nothing at all on the care of these animals. The figures and tables are clear, but some of them are rather small. The photomicrographs are uniformly poor in quality.

J. A. DUDGEON.

MITRAL VALVOTOMY

The Surgery of Mitral Stenosis. By Robert P. Glover, M.D., M.S.(Surg.), F.A.C.S., F.A.C.C., and Julio C. Davila, M.D., F.A.C.S., F.A.C.C. (Pp. 219+xii; illustrated. $9.50.) New York and London: Grune and Stratton. 1961.

Mitral valvotomy by the left atrial route was first performed in 1925 by Sir Henry Souttar. No progress with the operation was made after this single case until 1948, when Bailey, Glover, and O'Neill, and Sir Russell Brock published reports of successes. In the 12 years since then many hundreds of operations have been performed. Now Glover with J. C. Davila has produced from Philadelphia this valuable monograph, based on 1,000 cases, with an overall operative mortality of about 6%. Three-quarters of these patients have had lasting benefit. Tribute is paid to Souttar in the text, and in a foreword by Brock.

There is an excellent account of the anatomy and surgical pathology of the valve, with good diagrams and photographs, followed by an equally lucid description of the haemodynamics given by N. A. J. Hamer. Myocarditis as an important factor in chronic valvular disease is not restored to its former pre-eminence, but its existence is acknowledged. The book gains much by its brevity, but clinical description suffers; the electrocardiogram gets a short paragraph, and in indications for operation no more than a mention is given to systemic embolism. The surgeon hoping for help in the selection of patients with multiple valve lesions will find no easy answer here. A good account is given of the problem during pregnancy, but there is little about re-operation. The beautiful and detailed description of the operation reflects great experience and the benefits of a uniform technique. British surgeons will applaud the left-sided postero-lateral incision and the cautious approach to adequate division of commissures, with concentration on the anterior. Some may not agree that the carotid occlusion is of no value in preventing cerebral embolism, and many will be disappointed that there is no instruction in the use of the expanding dilator, but all who may be concerned will gain by reading this chapter. The section on results is confused by scattered figures and percentages relating to different groups of patients, which confusion is only slightly mitigated by diagrams in an appendix. The conclusion

that closed mitral valvotomy still has an important place in an era of open heart operations will be accepted for many years.

The stress laid on two principles in particular is welcomed: that the surgeon, while always a member of a comprehensive team, and aware that operation is no more than a part of a perhaps lifelong course of treatment, must make his own assessment and decision; and that the patient must always be kept fully informed.

M. MEREDITH BROWN.

TEXTBOOK OF GENERAL PATHOLOGY

Allgemeine Pathologie. Grundlagen und Probleme. By Dr. Erich Letterer. (Pp. 849+xx; illustrated. DM. 69.) Stuttgart: Georg Thieme Verlag. 1959.

In his new textbook of general pathology, the fruit of many years' labour, Professor Letterer has made a determined effort to build a logical system of theory from the multitude of empirical observations accumulated by generations of pathologists. Some parts of the structure may seem too ponderous for the meagre foundation of facts beneath them, but, viewed as a whole, his synthesis carries conviction. Lecturers tired of presenting their subject in the conventional way will find his book a useful source of new ideas and new viewpoints. It also contains valuable accounts of work hardly known in Britain, such as Ricker's concept of the "Endstrombahn"—the unit consisting of an arteriole together with the part of the capillary bed which it supplies—and its role in inflammation. Pathologists who read the specialist journals and are perplexed by strange phrases such as "serous inflammation," "Dyshorie," or "Relationspathologie" will find them explained here. One of Letterer's special interests is reflected in the masterly section on amyloid disease. The German is easy to read and the photographs are outstanding.

D. P. WINSTANLEY.

GARRISON-MORTON CITATIONS

Serial Publications Containing Medical Classics. An Index to Citations in Garrison-Morton. Compiled by Lee Ash. Foreword by Gertrude L. Annan. (Pp. 147+xxiv. $6.75.) New Haven: The Antiquarium. 1961.

Garrison and Morton's *A Medical Bibliography* is a classified and annotated list with admirable indexes of authors and subjects, but Mr. Morton did not judge it necessary to index the titles of the journals in which the individual papers cited first appeared. Mr. Lee Ash, of New Haven, Connecticut, has now done this in his *Serial Publications Containing Medical Classics.* The usefulness of this index will be best appreciated by librarians who are attempting to collect medical classics for their own institutions and by booksellers who specialize in "Garrison-Morton classics." An odd volume or number of a journal which might otherwise have been discarded without a second thought will now be checked against Mr. Lee's index to ensure that it does not contain one of the papers which, in the booksellers' hands, would be likely to become a lucrative article of commerce. A purely accidental outcome of this work, and one which has escaped comment by its author, is the interesting light which is thrown on the comparative status of the journals, judging by the number of classics which each contains. The scope is world-wide and includes all the greatest journals in the whole field of the medical sciences, yet the one which heads the list is the *British Medical Journal,* with 122 classic papers; second is the *Lancet,* with 117; third the *Journal of the American Medical Association,* with 102. Others, even the *Philosophical Transactions* (82) and the celebrated specialist journals, come a long way behind.

The index is preceded by Mr. Morton's Woodward Lecture delivered at Yale University on May 31, 1960, in which he gives "the story of the Garrison-Morton Bibliography."

F. N. L. POYNTER.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received.

Control of Ovulation. Proceedings of the Conference held at Endicott House, Dedham, Massachusetts, 1960. Edited by Claude A. Villee. (Pp. 251+x; illustrated. 70s.) Oxford, New York, London, Paris: Symposium Publications Division, Pergamon Press. 1961.

Textbook of Medical Treatment. By various authors. Edited by Sir Derrick Dunlop, B.A.(Oxon.), M.D., F.R.C.P.Ed., F.R.C.P.Lond., Sir Stanley Davidson, B.A., M.D., Hon.M.D. (Oslo), F.R.C.P.Ed., F.R.C.P.Lond., and S. Alstead, C.B.E., M.D., F.R.C.P.Ed., F.R.C.P.Lond., F.R.F.P.S. Eighth edition. (Pp. 983+xix. 60s.) Edinburgh and London: E. and S. Livingstone Ltd. 1961.

Radiographic Anatomy of the Human Skeleton. A Handbook for Radiographers. By W. H. Johnson, F.S.R., and J. A. Kennedy, M.B., M.R.C.S., D.M.R., D.M.R.D. (Pp. 279+viii; illustrated. 50s.) Edinburgh and London: E. and S. Livingstone Ltd. 1961.

Oakes' Pocket Medical Dictionary. Compiled by Nancy Roper, S.R.N., R.S.C.N., S.T.D.(Lond.). Ninth edition. (Pp. 491+xii. 8s. 6d.) Edinburgh and London: E. and S. Livingstone Ltd. 1961.

Proceedings of the Fourth International Congress on Clinical Chemistry, Edinburgh, 14th to 19th August, 1960. Edited by C. P. Stewart, P. C. Jocelyn, and S. C. Frazer. (Pp. 211+xvi; illustrated. 35s.) Edinburgh, London: E. and S. Livingstone Ltd. 1961.

Recurrent Dislocation of the Shoulder. By H. F. Moseley, M.A., D.M., M.Ch.(Oxon.), F.R.C.S.(Eng. and Canada), F.A.C.S. (Pp. 163+xii; illustrated. £6.) Edinburgh and London: E. and S. Livingstone Ltd. 1961.

13 Famous Patients. By Noah D. Fabricant, M.D. (Pp. 231+ix; illustrated. 30s.) Philadelphia, New York: Chilton Company. 1960.

Praktische Hämatologie. Differentialdiagnose, Therapie, Methodik. By Professor Dr. H. Begemann and Dr. H.-G. Harwerth. (Pp. 298+xii; illustrated. DM. 33.) Stuttgart: Georg Thieme Verlag. 1961.

Blood and Other Body Fluids. Analysis and Compilation by Philip L. Altman. Edited by Dorothy S. Dittmer. Prepared under the auspices of the Committee on Biological Handbooks. (Pp. 540+xvii. $10.) Washington: Federation of American Societies for Experimental Biology. 1961.

A Study of the Psychiatric Nurse. By Audrey L. John, B.Com.(Soc. Stud.), Ph.D., S.R.N., S.C.M. (Pp. 239+viii. 21s.) Edinburgh, London: E. and S. Livingstone Ltd. 1961.

Hospitals and Health Services in Britain and the United States of America. Some Reflections on Voluntary Effort for the Maintenance and Improvement of the Hospital and Health Services in Britain and on Financial Provision for Medical Care in Hospitals in the United States of America. By John Dodd (Pp. 148+v. 12s. 6d.) Bristol: British Hospitals Contributory Schemes Association. 1961.

Anatomy and Physiology. By Diana Clifford Kimber, Carolyn E. Gray, A.M., and Caroline E. Stackpole, M.A. Fourteenth edition by Lutie C. Leavell, M.A., M.S. (Pp. 779+x; illustrated. 48s. 6d.) New York: The Macmillan Company. 1961.

Proceedings of the Conference on Human Chromosomal Abnormalities. Edited by Professor William M. Davidson, M.D., and D. Robertson Smith, M.A., M.D. (Pp. 148; illustrated. 21s.) London: Staples Press. 1961.

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THE PLATT REPORT

The Platt Committee recommended that the reviews of medical staffing should be carried out by hospital boards with consultant advice. They should report to the Ministers with proposals for such additional consultant appointments that may be required and for posts in the medical assistant grade. Their reports should deal with the development of the firm system, the reorganization of the consultant service, and the development of rotational schemes of training for senior registrars, and the establishment of regional advisory committees on senior registrars. In short, boards should produce for the Minister a blueprint for putting the Platt Committee's recommendations into effect. The Ministers should then ask advice on the proposals for additional consultants and for the establishment of medical assistant posts from a professional committee consisting of members appointed by them and by the Joint Consultants Committee. The Report has been discussed by various committees, and the time for action on it must be approaching.

A measure of the discontent in the N.H.S. hospital system is provided by the fact that there were recently not enough applicants to fill the registrar vacancies in a famous London teaching hospital. What a contrast with the position that obtained before the war! For this reason alone we hope that action on the Platt Report on the staffing of hospitals will not be long delayed. The plight of the time-expired senior registrars, which was the presenting symptom of the problem that Sir Robert Platt and his Working Party were asked to investigate, is still as bad as ever, and a signal to go ahead would at least be a step towards stopping the rot. Sir Robert Platt, when he discussed his report with the Consultants and Specialists Committee recently,[1] said that the Working Party was unanimous in its view that more consultants were needed, and he was in no doubt that in making up the deficit promotion would come to the presently redundant senior registrars. In answer to those who thought the Working Party had done nothing about the senior registrar problem, Sir Robert said that, in his own view, "they had solved it completely." The recommendation that in future the number of senior registrar appointments should be kept more in line with expected consultant vacancies, though easier said than done, seems likely to ensure as much as anything can that the problem will not appear again in such an intractable form. The chairman of the Hospital Junior Staffs Group of the Association, Dr. Hamish Watson, has welcomed[2] most of the report, but among the points now at issue he mentioned the apparent lack of consideration for the present time-expired senior registrars.

The C.C. and S. Committee has also welcomed the Working Party's recognition of the inadequacy of present consultant establishments and its proposal that they should be reviewed as a preliminary to making additional consultant posts. At its last meeting[1] it recommended that this review should take place forthwith before any decisions were made on the action to be taken on the other recommendations of the Platt Report. Doubts have been expressed about the proposed medical assistant grade. The Platt Committee has made a good case for such a grade. It found that a surprising number of registrars continue to be employed in their posts indefinitely, and it contended that those doctors who wish for one reason or another to go on working in hospitals should be able to do so with security and on suitable terms. The Working Party also saw the medical assistant grade as a way of providing more opportunities for general practitioners to work in hospitals. The G.M.S. Committee, meeting on July 6 (see *Supplement*, p. 33), was on the whole sceptical about the proposed new grade. Dr. J. C. Cameron observed that now "the hospital service was breaking down attempts were being made to bring G.P.s back into it." Mr. Nicholson-Lailey, speaking for the C.C. and S. Committee, feared that the medical assistant grade might become another S.H.M.O. grade, and favoured the view that it should be limited in the first place to part-time practitioners working in the hospital service. The Hospital Junior Staffs Group, which represents all doctors below specialist rank, is opposed outright to having both registrars and medical assistants in the staffing structure.[2] The Working Party itself stated that the establishment of the medical assistant grade was dependent on a satisfactory standard of consultant staffing, and when the consultant staffing requirements are better known the present picture of what is needed in the way of assistants may change.

[1] *Brit. med. J., Suppl.*, 1961, 1, 290.
[2] Ibid., 1961, 1, 291.

N.H.S. CROSS-EXAMINED

"History has not infrequently been falsified by those seeking to explain, or indeed defend, great social changes by picturing them as the inevitable reactions against ancient and intolerable evils. The apologists of the National Health Service have been tempted to disparage what went on before and thus to exaggerate the degree to which we have profited from the change. It is an unfortunate, although probably inescapable, consequence of 'having medicine dragged into politics' that nearly everything connected with the subject comes to be discussed in terms of black and white." This quotation comes from the first of a series of booklets on the Health Service by John and Sylvia Jewkes.[1] As Professor Jewkes was a member of the Royal Commission on the Remuneration of Doctors and Dentists in the N.H.S., and, moreover, the author of a minority report which many thought showed a greater appreciation of the position of the medical profession than did the majority report of the Commission, these pamphlets should make their impact on a debate which still continues. In these days of committees that last two, three, or even more years before they can produce a report that really satisfies nobody, it is a treat to have the fresh opinions of independent minds. In their preface the Jewkes's say that the ways of providing medical services arouse more public passions than almost any other subject. In fact, politicians and spokesmen for the medical profession on both sides of the Atlantic rant about the merits of their own medical services and the demerits of the other in terms that are often painfully reminiscent of the two Mr. K.s talking about their political systems. And so the Jewkes's come in with a quiet voice to state some facts in the situation that could be read with benefit by medical men and medical politicians on both sides of the Atlantic. To quote the final words of their pamphlet: "There is something to be said for getting the record straight."

Looking at "The Genesis of the British National Health Service," to use the title of the pamphlet, the Jewkes's note the strong indications of radical thinking about the post-war arrangements of medical services in the draft interim report of the Medical Planning Commission set up by the B.M.A.[2] There can be little doubt that this report strongly influenced the Beveridge report on social security which appeared subsequently. The Jewkes's draw attention to the curious ambivalence there is in those who speak and write about the Health Service. They quote from a book[3] written in 1957 in which a young doctor refers to the "three closely-woven strands of frustration, fear and despondency" in British hospitals. (The author of that book could find his words borne out to-day by the fact referred to in the preceding leading article that a great London teaching hospital has no applicants for certain registrar vacancies.) And the same young doctor is quoted as going on to refer to the "breadth of vision which produced this unique organization." How, ask the authors of the pamphlet, can a system which creates despondency and frustration among doctors be fundamentally sound in conception? They quote Lord Cohen of Birkenhead, who in the House of Lords on November 19, 1959, referred to the perpetuation of the old antagonisms between the voluntary and municipal hospitals, to the isolation of the general practitioner from the hospitals, and to the filling of hospital beds according to Parkinson's Law. And then, on the other side, Lord Cohen goes on to say that he doubted whether any political measure (i.e., the N.H.S. Acts) had ever brought so much relief and hope to those who needed it. This is, of course, a more moderate attitude than that expressed by spokesmen for the Labour Party. As an example of this the Jewkes's quote Mr. Harold Wilson in the House of Commons nine months ago: "If the Tories laid their sacrilegious hands on the Health Service, which the Opposition regarded as the very temple of the nation's social security system, the Labour Party would fight it with the same determination which they had brought to fighting the Rent Act." This mystical flight is far removed from the prosaic statement made in 1945 by the late Mr. Aneurin Bevan, which appears opposite the title page of this pamphlet: '"If we were rich enough we would not want to have free medical services, we could pay the doctor."

Professor and Mrs. Jewkes make some most interesting comparisons between the medical services available in Britain and those available in the U.S.A. They are interesting because medical men on both sides of the Atlantic tend to cock snooks at each other in this matter. The Jewkes's give facts and figures to show that before the war the U.S.A. "was spending in total on medical care a somewhat larger percentage of its national income than was Britain." They go on to say that "since 1949 the proportion in the United States has, and in Great Britain has not, increased." The gap is now wider than it was. They note, too, that the quantum of medical care has grown more rapidly in the U.S.A. than in Britain. Looking at hospital facilities, they

[1] *The Genesis of the British National Health Service*, 1961. Basil Blackwell. Price 6s. 6d. or $1 net.
[2] *Brit. med. J.*, 1942, **1**, 743.
[3] Goldman, Louis, *Angry Young Doctor*, 1957.

conclude that it is doubtful whether in proportion to the population there were more general hospital beds in 1959 than in 1948 or indeed than in 1939. They draw attention to something which so many have conveniently forgotten, and that is the expansion in hospital facilities that followed the Local Government Act of 1929. And they point out that in 1939 Great Britain was more amply supplied with hospital beds than the U.S.A.; but now the trend is reversed. They conclude as a result of their examination of the position, backed up by informative tables in the appendix to the pamphlet, that "the average American now has more medical services than the average Briton and that the gap between the two has been widening."

The Jewkes's expose once again the fallacies so misleadingly put before the public after the war that the poor sections of the population suffered from lack of medical care. They make an observation that Sir William Osler made many decades ago that the people who had the biggest grievance in respect of medical services were the middle classes. Many authorities are quoted to support the view that the National Health Service suffers from the complexity and inflexibility of its administration. The Guillebaud Committee concluded that "there is a real danger of the administrative side of the National Health Service getting bogged down in a morass of committees." The present Minister of Health ten months ago said: "The great machine is bound to have a one-track mind, to be cumbrous and unresponsive, to abhor variations, to be insensitive to the world around it."

The above are just some points brought out in a highly informative, cogently argued pamphlet, which will give the reader a taste for the other ones that the Jewkes's promise to provide. Perhaps one of their most challenging observations is the suggestion that the National Health Service may have "positively hindered the growth of British medical services." We may or may not agree with this, but at least the remark comes as a challenge to those who, with undue cause for self-congratulation, boost Britain's N.H.S. as if it were one of the most remarkable things that has happened in the twentieth century. The same unctuous self-praise—that the N.H.S. is an example to the world—may well be, as according to the Jewkes's foreign observers say about us, "the kind of pretentious claim likely to be indulged in by a power which finds its place in the world slipping, does not relish it, and seeks compensation in national day-dreaming."

COMPREHENSIVE ACCIDENT SERVICES

The Accident Services Review Committee of Great Britain and Ireland[1] has just published its first report[2] after a little more than a year's deliberation. That agreement on the report has been reached between members representing so many interests (16 organizations in all) is highly creditable. The committee is a professional body "primarily concerned with laying down principles for the type of service required . . . and to keep under continuing review the accident services of Great Britain and Ireland." Its chairman is Mr. H. Osmond-Clarke, one of the members representing the British Medical Association. If the report is a portent of the will of clinicians to mould the pattern of our hospitals for the better, then action on the committee's recommendations should swiftly follow.

For the first time injuries from accidents have been accepted as the concern of all branches of medicine, with responsibilities to be shared between the general practitioners, the industrial medical services, and the hospitals, according to the facilities that are now, or can in the future, be provided. The report makes the important point that only certain hospitals should be designated as "accident units," and these should be supported by a "peripheral casualty service." In no other way can such hospitals be equipped and staffed to cover their essential function of providing a service for 24 hours a day. The committee calls for a closely co-ordinated regional accident service in a three-tier scheme, to be developed around a headquarters to be called the "central accident unit," which should usually be attached to a teaching hospital. This is not a new conception, but is based on the working experience of such centres in Birmingham, Oxford, and Sheffield.

The need for a new organization for the care of the injured has long been recognized. As far back as 1912 a committee of the B.M.A. published a report entitled "The Treatment of Simple Fractures." Other reports have appeared at regular intervals. Yet a survey on casualty services published in 1960 by the Nuffield Provincial Hospitals Trust[3] contained the general criticism that even now "casualty departments' supporting services are inadequate or frequently non-existent and their relationships with other special departments are not close enough." And more recently still the Platt Report[4] found the medical

[1] See annotation, *Brit. med. J.*, 1960, **1**, 792.
[2] Accident Services Review Committee of Great Britain and Ireland. Interim Report, 1961. Obtainable from the Secretary of the Committee, B.M.A. House, Tavistock Square, London W.C.1.
[3] *Casualty Services and their Setting—A Study of Medical Care.* Published for the Nuffield Provincial Hospitals Trust by the Oxford University Press, 1960. See leading article, *Brit. med. J.*, 1960, **2**, 920.
[4] Medical Staffing Structure in the Hospital Service. Report of the Joint Working Party, 1961. H.M.S.O., London. See leading article, *Brit. med. J.*, 1961, **1**, 883.
[5] Association of Hospital Management Committees. Annual Report for the Year 1960-61.

staffing of casualty departments "the least satisfactory feature in the present hospital staffing system." Only 25% of 140 hospital management committees asked for their views on the adequacy of their arrangements for the reception and treatment of casualties expressed satisfaction with the present state of affairs: many were deeply concerned at the difficulty, if not impossibility, of discharging their responsibilities.[5]

The new report sets out to remedy these long-standing defects. It states that injuries from accidents "have become one of the most important medical problems." Man was ever prone to injuries, and surgery was founded for their treatment. But from the beginning of this century powered vehicles have changed the nature and severity of injuries and added a new factor—a multiplicity of severe injuries in one victim. As a result trauma now cuts across all branches of medicine with a complete disregard of the man-made boundaries of the different specialties and has introduced a new urgency in treatment. From the evidence of the second table in the report, road casualties have risen from 138,774 in 1926 to the record figure of 333,453 in 1959. Since accidents are directly related to the numbers of vehicles on our roads, by the end of this decade the total of road casualties will certainly be higher still. This is the main challenge to quick action in the establishment of a national accident treatment service.

The report, perhaps wisely, has concerned itself more with the training of surgeons of the future than with allocating detailed responsibilities now for the overall care of victims of multiple injuries. Yet so quickly progressive is the "illness of trauma" that as soon as the severely injured are admitted to hospital responsibility should be centred on one surgeon. There must be unified control of resuscitation, diagnosis, and treatment, leaving the surgeon in charge to secure such assistance as he may deem necessary within the time that treatment has the best chance of success.

The report reproduces plans of existing accident units. The Birmingham Accident Hospital is shown as having a separate department for the reception of the seriously injured. There the duty surgeon immediately accepts full responsibility for the care of each patient on admission. It would be informative if to each of the plans shown a date was added, giving the age of the buildings in which they are situated. New buildings with every modern amenity based on the experience of these pilot schemes could well be the first stage of development of a national accident service. No one can now doubt the need for this on medical and financial grounds. The present cost of accidents to the community as outlined in the report is a matter of grave national concern. In the committee's view, only by the establishment of a comprehensive accident service for the whole country can "the present appalling waste of life, disability, and unnecessary suffering which are caused by accidents be reduced."

JAUNDICE AND DEAFNESS

The incidence of serious perceptive deafness as a result of jaundice in the newborn baby is commoner than is generally realized. Drs. L. Fisch and A. P. Norman, in a paper published in this issue, report six cases of typical perceptive partial deafness in 50 children jaundiced at birth. Thirty years ago neonatal jaundice was regarded as a harmless condition except when due to congenital obliteration of the bile ducts, though J. Orth,[1] in 1875, had noted the yellow pigmentation of the basal ganglia which was given the name "kernicterus" by G. Schmorl[2] in 1904.

Our present understanding of the brain damage which neonatal jaundice can cause dates from the discovery of the rhesus factor in 1940 by K. Landsteiner and A. S. Wiener[3]—and the recognition of this as the cause of haemolytic disease in the newborn. Later it was realized that neonatal jaundice and kernicterus led to the development of spasticity and athetosis. In 1948 L. Lande[4] first noted that deafness was part of the syndrome. In 1950 several papers appeared on the association of symmetrical high-tone deafness with athetosis, and four years later Florence Cavanagh[5] drew attention to the reduction in the incidence of perceptive deafness in a group of infants with erythroblastosis foetalis treated early by transfusion compared with babies treated late in the illness or not at all.

This partial elucidation of the problem of neonatal jaundice and rhesus incompatibility stimulated interest in the premature baby, who is particularly liable to become jaundiced, and it was found that these infants also develop spasticity, athetosis, and deafness. The treatment of the jaundiced "rhesus" baby is by exchange transfusion—the need for this procedure being determined by the level of haemoglobin, by the level of bilirubin in the cord blood, and by an assessment of the clinical condition. In the premature baby, if there is no iso-immunization, there is no fall in haemoglobin, but the high level of bilirubin is due to the immaturity of the liver. (Normal full-term infants have a bilirubin level at birth of 1.8 mg. per 100 ml., which rises over the next few days to 7–12 mg. and returns to less than 2 mg. per 100 ml. at the end of the first week.) The degree of bilirubinaemia which can be regarded as safe, and the level which may

[1] Orth, J., *Virchows Arch. path. Anat.*, 1875, **63**, 447.
[2] Schmorl, G., *Verh. dtsch. path. Ges.*, 1904, **15**, 109.
[3] Landsteiner, K., and Wiener, A. S., *Proc. Soc. exp. Biol. (N.Y.)*, 1940, **43**, 223.
[4] Lande, L., *J. Pediat.*, 1948, **32**, 693.
[5] Cavanagh, F., *J. Laryng.*, 1954, **68**, 444.
[6] Trolle, D., *Acta paediat. (Uppsala)*, 1961, **50**, 392.
[7] Zuelzer, W. W., and Brown, A. K., *Amer. J. Dis. Child.*, 1961, **101**, 87.
[8] Day, R. L., *Pediat. Clin. N. Amer.*, 1961, **8**, 539.

indicate the need for exchange transfusion, are therefore extremely important. Most centres nowadays try to prevent the bilirubin rising above 18 or 20 mg. per 100 ml.

The only method of controlling the bilirubinaemia at present is by exchange transfusion. Injections of albumin have been used experimentally to lower the bilirubin in jaundiced rats, but these investigations are not yet advanced enough for application to clinical medicine. Many premature babies have suffered from anoxia at birth and their general condition is poor—quite apart from the problem of jaundice. There is some danger to them in an exchange transfusion. According to Dyre Trolle[6] the risk of death from transfusion is four times greater than the risk of athetosis.

W. W. Zuelzer and A. K. Brown[7] mention one baby with a bilirubin level of 54 mg. per 100 ml. who developed normally, whereas R. L. Day[8] cites a case of kernicterus occurring in a baby whose bilirubin never rose above 12.2 mg. per 100 ml. Many workers are trying to clarify the relationship between bilirubinaemia and the neurological sequelae, of which perceptive deafness is one. It appears likely that other factors are involved. Fisch and Norman mention the suspicion which has fallen on vitamin K and note that the incidence of deafness appears to have been reduced since a smaller dosage of the vitamin was started in 1954. A single dose of 1 mg. is now usual in many centres, though this is occasionally repeated. Anything damaging the liver—the immature liver—of these small babies may add to the risk. Certain sulphonamides are toxic to the liver of the newborn infant, and some drugs given to the mother in late pregnancy may affect the foetal liver—for instance, hypotensive and antimalarial drugs and tranquillizers.

Many questions remain unanswered, and there is no clear guide to the treatment of jaundiced premature babies. It is, however, well established that such children may suffer from deafness, and the earlier this deafness is detected the better it will be for the child. His deafness cannot be cured, but he (and his parents) can be taught how to use a hearing-aid and how to speak correctly, and he can receive special educational help. This type of high-tone deafness is unlikely to be suspected by parents. Such a child hears low frequencies normally: his difficulty is with certain consonants, and he is often taken to the doctor because he has a speech defect. Arrangements can now be made in most areas for a baby's hearing to be tested at a welfare clinic or at the otological department of a hospital.

TRENDS IN JUVENILE DELINQUENCY

The World Health Organization has for some years taken an active interest in the problems of juvenile delinquency and has recently sponsored a study of them by T. C. N. Gibbens.[1] Different standards and procedures make international comparisons difficult; and the extent to which delinquency is reported to the police or observed by them depends on several factors. The most important changes affecting the statistics of prevalence are the changes in the definition of delinquency. It has long been known that young people convicted come mainly from the less privileged sections of the community and that they are largely concentrated in certain areas of cities; experience has shown that it may be many years before the rehousing of slum dwellers reduces delinquency among them. Perhaps the most promising trend of the past decade has been towards closer co-operation between sociologists and psychiatrists in their approach to these questions. R. L. Jenkins[2] distinguished four categories of delinquent: (1) the situational category (17%), containing those whose personality was essentially normal and who hardly needed training-school treatment; (2) the pseudo-social category (14%), containing those who were "socialized" within a delinquent group; (3) the personality category (64%), containing those in whom the pattern of delinquent behaviour had become in some way internally rooted; and (4) the asocial group, containing the most seriously disturbed, likely to become adult asocial and amoral psychopaths. J. Rich[3] similarly attempted to differentiate types of stealing—"marauding" offences, with three or more boys stealing when opportunity presented, but without planning; "proving" offences, committed usually alone with the object of demonstrating one's manhood, and so on.

Gibbens's survey shows that in Denmark there has been of recent years little change in juvenile crime; in France and the United Kingdom it has fluctuated, and in the last three years has shown a rapid increase after a previous fall. In the United Kingdom there has been no great increase in offences committed by boys between the ages of 8 and 13, despite the considerable population increase at these ages. A sharp increase in juvenile delinquency is not, on the whole, characteristic of those countries (Israel, Yugoslavia, Poland) which are undergoing the most rapid social reconstruction or development. So far as delinquency is concerned, urban society appears to have four main characteristics—the greater complexity of its economic and social organizations, its increasing mechanization and standardization, the increased secularization of life, and the substitution of impersonal relations for personal as a result of those features. The most important aspects of urbanization in relation to juvenile crime are probably those which affect the quality of the family life of the child, or, in the second decade of life, the effects of delinquent subcultures. In older age groups, between 15 and 21, the pressure of economic changes merits further attention—the large increase in the earnings and spending power of teenagers, of which it has been estimated that about half goes in "entertainment," in the widest sense. Pressure to sell to this group often aims at persuading teenagers that they form a separate culture, neither children nor adults. There seems to be no reason to alter L. Bovet's[4] opinion, expressed ten years ago, that television and radio may determine the

[1] Gibbens, T. C. N., *Trends in Juvenile Delinquency*. World Health Organization, Geneva, Public Health Papers, 5, 1961.
[2] Jenkins, R. L., in *Social Work in the Current Scene*, selected papers, 76th Annual Meeting, National Conference of Social Work, Cleveland, Ohio, June 12–17, 1949. New York. 1950.
[3] Rich, J., *Lancet*, 1956, 1, 496.
[4] Bovet, L., *Psychiatric Aspects of Juvenile Delinquency*. World Health Organization, Geneva. Monograph Series No. 1, 1951.

content of offences but do not motivate them. Motor-car stealing has shown a great increase; it is estimated that in London 1% of the available cars are stolen or "borrowed" every year. Other crimes that have increased are shop-lifting, fruit stealing (especially in Israel), and, rather surprisingly in Yugoslavia and some other countries, "ticket touting," where youths apparently buy up all the seats in a cinema or theatre and sell them at a profit. Some types of offence seem to show a double standard of morality, since those responsible may never have committed any other crimes and seem to be generally law-abiding; moreover, the parents may appear to condone them. It appears that public property is viewed in a different light from private property and that, with the growth in public ownership, it is increasingly regarded as fair to steal from the State, as in the frequent offence of not paying for one's railway ticket, which accounts for a large proportion of offences in some juvenile courts.

Gibbens concludes that in clinical experience the problems of delinquency arise in much the same way to-day as yesterday, from serious deprivation and major disorders of family life. Discussing trends of prevention, he refers to the many attempts that have been made to deal with pre-delinquents in their natural groups, attempts dogged by the major difficulty of "reaching the unreachable," for the clubs and services provided tend to be used by those who need them least. In both preventive and treatment programmes there is a growing tendency to design experiments in such a way that some evaluation can be made of success or failure, and it is suggested that in future it may prove more useful to study the results of those forms of prevention and treatment which are at present in use, without regard to causal explanations, and on the basis of this evaluation to suggest improvements in the use of existing facilities. "The more carefully those evaluative studies have been made, the more disconcerting they have been to established theories in both psycho-pathology and social work; they can thus hardly fail to give rise to new orientations."

POISON ON THE LAND

A few months ago we drew attention to a report[1] which suggested, without firmly proving, that wild birds were dying as a result of ingesting certain chemicals used in agriculture for the dressing of seed. One of the two interim reports[2] issued recently from the Infestation Control Laboratory of the Ministry of Agriculture states that an investigation carried out in order to determine the cause of death of wood pigeons believed to have been poisoned showed that "there is no room for doubt that the birds [the flesh of which was found on analysis to contain toxic chemicals] obtained these chemicals from consuming seeds dressed with aldrin, dieldrin, or heptachlor. The Minister of Agriculture, Mr. Christopher Soames, has therefore done well to arrange for restrictions on the use of these three insecticides. But it is clear from a number of unanswered questions in the two interim reports on which this decision is based that this is only an initial step forward and that much more must be done to ensure that all potentially toxic chemicals used on the land are rigorously screened by independent investigators before and not after they are placed on the market. The reports by the staff of the Ministry's Infestation Control Laboratory show that what the Minister called "widespread" casualties among birds since the spring of 1956 can be attributed to a great extent to the equally widespread use of some dual-purpose seed dressings. These are seeds dressed with a combined fungicide (usually based on mercury) and an insecticide in the form of one of the chlorinated hydrocarbons. Chemical analyses of the organs and flesh of some of the birds found dead disclosed up to 38 parts per million of dieldrin and 22 p.p.m. of heptachlor. One of the reports states that the hazard to man of eating birds which have fed on dressed seeds is not serious because of the "impossibly" large amounts of flesh which would have to be eaten, but the other report remarks that it is "almost certain" that many predatory animals (foxes, badgers, cats, and dogs) died through eating poisoned birds.

As he announced in Parliament on July 3, the Minister has now agreed to restrict the use of aldrin, dieldrin, and heptachlor to occasions when there is a demonstrable risk to autumn- or winter-sown grain from depredation by a chronic pest, the wheat bulb-fly. They will not be used for the spring sowing, the time when most of the recent damage to bird and mammalian life has been done. It is likely that most manufacturers who supply the raw materials for seed dressings, about the efficacy of which there is no argument, will substitute a less lethal chlorinated hydrocarbon called lindane or B.H.C. (benzene hexachloride) for those under the restrictions now agreed upon.

It is greatly to the credit of the voluntary protection societies that they have been so prompt in showing how destructive certain classes of seed dressings may be to wild life. But the side-effects of an arsenal of pesticidal sprays, washes, dusts, and smokes remains almost entirely uninvestigated. In the last ten years, British agriculture has been revolutionized by the farmers' ability to produce a succession of cash crops instead of the periodic planting of fodder for stock. This means a continual call on the chemical manufacturer, whose ingenuity has not been matched by ecological research at the academic level. The difficulty was summed up in the last annual report (1959–60) of the Advisory Council on Scientific Policy: "There is no clear general responsibility for the conservation of our natural resources whether of minerals, water, land use, biological resources or fisheries; and the responsibilities of the Nature Conservancy, and of other Departments concerned, cover only limited aspects."

[1] *The Deaths of Birds and Mammals connected with Toxic Chemicals in the first half of 1960*. Royal Society for the Protection of Birds, 25 Eccleston Square, London S.W.1. See annotation, *Brit. med. J.*, 1961, 1, 651.

[2] Ministry of Agriculture, Fisheries, and Food. *Deaths Among Wild Creatures from the Consumption of Dressed Seeds*. Appendix A, *A Summary of Recent Investigations*: Appendix B, *A Summary of the Present Position*.

SMALLPOX IN CENTRAL PROVINCE, NYASALAND

BY

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Smallpox has always been endemic in Nyasaland, with occasional outbreaks of such proportions that they can be termed epidemics.

Vaccination campaigns are continually under way, and the year 1960 was no exception. In the middle of the year, however, and especially in the Central Province, much political opposition was encountered. As a result the vaccination programme was greatly restricted, and in some villages was brought to a standstill. This opposition was withdrawn in October and the vaccination campaign was reintroduced, though by this time it was too late to prevent outbreaks of the disease.

Isolation

The traditional method of isolation for the odd case of smallpox is to build a grass hut away from the village in an attempt to keep the patient in strict seclusion, prohibiting all visitors. Towards the end of October and the beginning of November the number of known cases occurring around the Lilongwe Township increased so much that other means of isolation had to be sought. Accordingly a large and spacious building, formerly used by the Public Works Department as a brick and tile works, was taken over. The building is approximately five miles (8 km.) from the Lilongwe Township, near enough for adequate supervision by the hospital staff. The main hall measures 93 by 48 ft. (28.3 by 14.6 m.) and on each side there is a walled and roofed verandah. The building is constructed of latticed brickwork with a corrugated iron roof, and has an equable temperature even in the hottest weather. Temporary kitchens and latrines were brought into use and water was supplied by means of a prime moving 800-gallon (3,630-litre) tanker.

Admissions

Despite the renewed efforts at mass vaccination there was an appreciable build-up in admissions over the first two months (November and December), with a slight fall over the Christmas season, and then a further increase. During the five-month period under review the admissions numbered 620 and the deaths 95 (see Table). Cases of variola minor accounted for roughly 70% of the total, and there were nine cases of vaccinia. Six patients had confluent smallpox and four of them died. It is impossible to estimate the number of patients and the number of deaths that were concealed, but from reports received these must have been considerable.

Age	No. Admitted				Deaths			
	Males	Females	Total		Males	Females	Total	
			No.	%			No.	%
Under 1 yr.	60	71	131	21·1	18	6	24	25·2
1 year	80	91	171	27·6	5	16	21	22·1
2–4 yrs.	92	83	175	28·4	11	8	19	20·0
5–9 ,,	41	44	85	13·7	5	11	16	16·8
10–18 yrs.	7	15	22	3·5	7	5	12	12·6
Adults	8	28	36	5·8	1	2	3	3·1
Total	288	332	620	100·1	47	48	95	99·8

Diagnosis and Treatment

Diagnosis did not present any great problem. The distribution of most of the rashes was typical (Figs. 1 and 2), but it is surprising how many of the cases showed pocks on the palms and the soles.

The history of onset was usually fever and general weakness for from three to five days and then the appearance of the rash. The source of infection was

FIG. 1

FIG. 2

FIG. 3

FIG. 1.—Early healing stage in variola major, showing scars. FIG. 2.—Variola major, almost confluent. FIG. 3.—Ophthalmia right eye due to pocks.

usually attributed to a neighbouring village or was stoutly alleged to be "unknown." Owing to the fears and superstitions of the relatives it was often difficult to elicit a proper history.

So far as the smallpox was concerned controlled experiments were made with various antibiotics, but none of them shortened the course of the disease to any appreciable extent. Calamine lotion was helpful in relieving local irritation, and in preventing extensive scratching which might have led to local sepsis.

Many of the patients developed conjunctivitis, which responded to chlortetracycline eye ointment. Five children developed pocks on the conjunctiva (Fig. 3); one child lost the sight of both eyes, three lost the sight of one eye, while the fifth child recovered completely. The most common complication, occurring mainly in the two younger age-groups, was bronchopneumonia, and this responded favourably to procaine penicillin.

The children were often undernourished, and various vitamin compounds with adequate diets helped considerably. If malaria developed it was treated with chloroquine sulphate.

If the patient survived the first three days of the toxaemic stage the prognosis was usually good, but it is impossible to be dogmatic on this point.

Vaccination Programme

Teams of vaccinators were organized in a concentrated drive to vaccinate the whole population. These teams were under the supervision of health inspectors and field officers, with trained health assistants and lay vaccinators. Vaccination points were established at which occupants of near-by villages could attend on specific days at fixed times. Individual villages were visited in an attempt to find stragglers. The people soon realized that these measures were designed for their benefit and little opposition was encountered. Despite these all-out efforts, however, cases of smallpox were still occurring at the time of writing.

Nothing was discovered which proved to be specific against smallpox as such, but prompt treatment of intercurrent infection and other complications did much to keep down the number of deaths.

Summary

Smallpox reached what may be termed epidemic proportions in the Central Province of Nyasaland in the third quarter of 1960. This could be attributed to political opposition to vaccination campaigns.

Traditional methods of isolation were abandoned, and what became virtually an isolation hospital for smallpox alone was opened near Central Province Headquarters at Lilongwe.

Opposition to vaccination ceasing in October, the programme could proceed, but by this time the damage was done.

Diagnosis presented no difficulty, but experimental treatment itself produced no new discovery, success being confined to complications.

As the number of cases increased, so the population became more and more co-operative towards the intensified vaccination drive. The majority of cases occurred in children under 10 years of age with no significant sex differentiation. Above this age female cases preponderated.

As regards death rates, the figures show a peculiar alternation of sexes in the four youngest groups. The greatest percentage of deaths occurred in children under 10 years of age.

I am grateful to Mr. Madoc-Jones, Lilongwe, who supplied the photographs, and to the Regional Director of Medical Services, Blantyre, Nyasaland, for permission to submit this article for publication.

A HOSPITAL PLAN BASED ON A TEACHING CENTRE

BY

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The Board of Governors of the United Birmingham Hospitals, in co-operation with the Regional Hospital Board and Birmingham University, is preparing a plan for the Teaching Centre.* This article describes the principles on which the plan is based.

In many respects the present position is a difficult one for hospital planners. After a prolonged period of relative inactivity there is to be a new phase of building. But although this is badly needed we are not yet ready for it: we have a great deal to learn about how a hospital should be built and how it should function. Enough is known to make us dissatisfied with what we have had in the past, but not enough to enable us to plan confidently for the future.

Confronted with these difficulties planners may be said to fall into three classes. There are some who see no reason to do more than modernize the fittings and improve the décor of the traditional hospital. Others see many reasons for change but feel that the difficulties are too formidable. Still others believe that, whatever the difficulties, they must be faced, since it is unthinkable that we should perpetuate a tradition which is manifestly incapable of solving problems which past successes and an enlarged concept of public responsibility have jointly created.

Some Outstanding Issues

The tenability of this last viewpoint turns upon whether the problems are serious enough to justify an attempt to find a radical solution. In general terms the main task confronting the Health Service is to restrict its cost while extending its usefulness, but such a statement is too vague for the present purpose. More specifically, some of the outstanding issues with which the traditional services have proved inadequate, and in relation to which future planning must be directed, are the following.

1. *Standard of Care of the Mentally Ill and Aged Sick.*—The system of care which has evolved during the past two centuries has been unable to provide an adequate standard of care for the mentally ill and aged sick. In view of the age trend of the population this problem cannot fail to become more serious, and our success in dealing with it will provide perhaps the best single test of the achievement of the medical services.

*The research for this investigation is being done by the Bartlett School of Architecture, University College, London, and the Department of Social Medicine, Birmingham University. It is supported by a grant from the Nuffield Provincial Hospitals Trust.

2. *Economy of Construction and Operation.*—So long as they are separate, general, mental, and chronic hospitals cannot be built or operated economically. This is because their patients are heterogeneous in respect of their need for care, the difficulties being analogous to those which would confront an industry which attempted to combine within the same building the making of plastics, the assembly of motor-cars, and the baking of bread.

3. *Flexibility.*—The traditional hospital is inherently inflexible, and can be made only a little less so by modifications of internal design. This is attributable basically to the permanence of construction inevitable in the single self-contained hospitals and to the separation of major classes of patients which prohibits conversion of unwanted facilities to alternative use.

4. *Unification of Services.*—Although the disadvantages of divided control are self-evident, the long-standing divorce of hospital from domiciliary care, and of preventive from curative medicine, stands in the way of a unified service.

Basis for Planning

It was suggested that if these and some other difficulties are to be overcome it will be necessary to eliminate two features of the hospital tradition: the separation of the mentally ill and chronic sick from the populations of general hospitals; and the concept of the hospital as a single independent building (McKeown, 1958). Instead, hospital planning might be based on centres with the following features: (1) The care of all types of patients at a common site. (2) Multiple buildings of varied size, design, and permanence of structure. (3) Classification of patients strictly according to need, and their location in facilities appropriate to their requirements. (4) Unification of the various phases of care—acute, rehabilitative, static, out-patient, and day—through a common medical and nursing staff. (5) Removal of restrictions which sharply divide domiciliary from institutional care, and hence prohibit the evolution of a unified service.

In part these proposals are an extension of ideas which are beginning to be widely accepted. For example, it is recognized that large isolated mental and chronic hospitals are undesirable, and that both psychiatric and geriatric facilities should be included in general hospitals. There is also considerable support for the view that a place should be found for general practitioners in hospital and for consultants outside it. And there is evidence of awareness of the advantages of multiple interdependent buildings in recent hospital developments at Glostrup in Denmark and of a common staff in Toronto in Canada. But these trends are no more than a tentative beginning, and their significance for future planning has not been fully appreciated. It was in an attempt to provide a comprehensive approach that the concept of a balanced hospital community was suggested.

But, whatever the merits of this concept, its impact on hospital planning must be limited until it has been examined in a real situation. Indeed, the proposals themselves are open to the criticism that they are capable of more than one interpretation. For example, what is meant by a common staff? Is it suggested that all doctors and nurses should have responsibilities for all phases of care? Would it affect surgeons as well as physicians, or only psychiatrists and geriatricians concerned with complex services? Again, it is one thing to propose unification of institutional and domiciliary services, and quite another, as experience of more than one national committee has indicated, to show how in the face of multiple and often conflicting interests it can be done. These and other questions clearly made it desirable to examine the proposals in relation to an actual site and a defined population.

The Birmingham Plan

The Birmingham Plan is based on a 60-acre site adjacent to the Queen Elizabeth Hospital and Medical School (A and B in Fig. 1). This is part of a larger area given in 1934 by Cadbury Brothers Limited to the City of Birmingham for construction of hospitals and playing-fields. Except for a laundry and power-house serving the hospital the land is undeveloped. There seems little doubt that, had it been possible to fulfil the intentions of the gift soon after it was made, the result would have been a hospital centre as conceived in the 'thirties: a group of independent and separately staffed hospitals on the same site, as exemplified at the Columbia and Cornell Medical Schools in New York. The delay has made it possible to consider the use of the land in the light of post-war experience of hospital building and the proposals for a balanced hospital community.

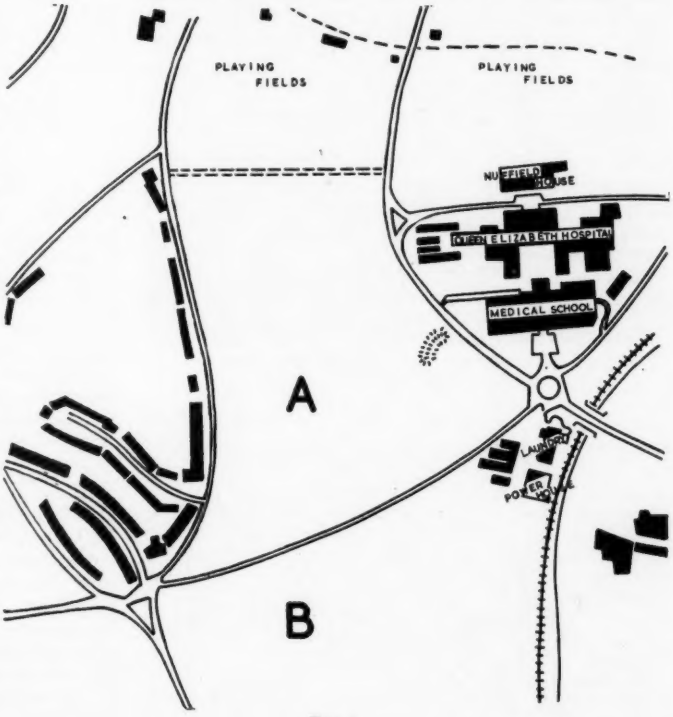


FIG. 1

These proposals were first made, however, in relation to hospitals in general, and it was clearly necessary to consider to what extent they are applicable at a teaching centre Teaching hospitals have been concerned with selected patients, and, almost exclusively, with institutional care, and the inclusion of a representative service requires substantial justification. Three reasons were suggested (McKeown, 1961). (1) It would give students a balanced view of the medical task, and of the best ways in which it can be tackled. (2) It would enable teaching centres, with their great assets in staff and resources, to contribute more to thought about the future of medicine which is inseparable from the future of its services. (3) It would make available to research workers what has hitherto been conspicuously lacking, a representative sample of patients drawn from an identifiable population.

Preservation of Traditional Interests

But if this programme is to be accepted it must be implemented without prejudice to traditional interests. For teaching and research purposes teaching hospitals will wish to continue to have access to highly selected patients derived from wide areas in which the related populations are unknown. Both interests can be accommodated by making the number of representative patients reasonable in relation to the number of selected patients. The way in which this can be done can be illustrated by reference to plans for the Birmingham Centre.

It is proposed that the teaching centre should accept responsibility for all institutional medical services for a population of approximately 100,000 in three city wards surrounding the centre. These wards were chosen because they are close, and because admission rates from them are already as high or higher than from any other parts of the city. The size of the population is admittedly arbitrary. It was intended to be large enough to provide more than token numbers of patients, and not so large that it would seriously affect numbers of selected patients drawn from other parts of the town, and from outside the town, for traditional purposes.

FIG. 2

Some explanation is needed of what is understood by a defined area: (1) It defines, and sets limits to, the size of the population for which the teaching centre should attempt to provide all hospital services. (2) It is the area within which close association with general practitioner and local authority services would be sought in an attempt to integrate domiciliary and institutional care. (3) No attempt would be made to draw patients strictly from the defined area. However, the association with general practitioners, as well as the personal convenience of patients, would lead to increasing use of the centre by patients in the area.

By this means it is suggested that the centre might cater broadly for the two classes of patients shown in Fig. 2. It would admit the selected patients, drawn from a wide area, who have represented almost an exclusive interest of teaching hospitals in the past. And it would admit all patients (interpreted as above) from the defined area determined by the boundaries of three city wards. It is upon the balance between these two activities that acceptance of the new responsibility without prejudice to traditional interests depends.

REFERENCES

McKeown, T. (1958). *Lancet*, **1**, 701.
—— (1961). Ibid., **2**, 1.

PAEDIATRICS AND PSYCHIATRY

[FROM A SPECIAL CORRESPONDENT]

On July 1 the Mental Health Committee of the British Paediatric Association, with the assistance of the Association for Child Psychology and Psychiatry, held a meeting in London to discuss recent progress of common interest in paediatrics and psychiatry. Several points about this meeting would have provoked surprise even a few years ago. First, that it was held at all; second, that close on 200 people (paediatricians, psychiatrists, and others from related disciplines) attended; third, that everybody spoke the same non-technical medical language. The chairmen of the three sessions were Professor A. V. NEALE, Dr. D. W. WINNICOTT, and Dr. R. MAC KEITH.

In an introductory talk Professor D. V. HUBBLE sketched the changing social patterns in this country. The character of the work done by doctors was changing correspondingly, and Professor Hubble emphasized their responsibility in adapting to new and developing situations. Many of the problems within the ill-defined boundaries of child psychiatry could and should, he said, be dealt with by paediatricians (and by general practitioners). But they would do this more confidently if the training facilities in university departments of child health were expanded. The need for adequate training at the undergraduate stage was affirmed by many speakers later.

New Ways in Paediatric Clinics

Under the title "New Ways in Paediatric Clinics" Dr DAVID MORRIS, Dr. DAVID WILLIAMSON, and Dr. JOHN APLEY described combined clinics, held by a paediatrician with a psychiatrist or with a psychiatric social worker. These interesting pioneering efforts provoked considerable discussion, largely concerning the roles of the personnel

concerned, and it was generally agreed that similar experiments should be widely encouraged. A psychiatrist had been asked to comment, and to make his points Dr. MICHAEL BALINT discussed a case, a child with asthma. He emphasized the intricacy of the many factors that preceded the appearance of overt disturbances, and suggested that the child, and not only the mother, should take an active part in the consultation. He asked: What does the symptom mean? Which member of the family is really ill? Who should treat whom?

An informal duologue between Dr. MILDRED CREAK and Dr. MAC KEITH raised some friendly controversial issues. Why did not child psychiatrists concentrate on children under 5 years of age? Why were the fathers of disturbed children not brought into the picture more often? The difficulties of "control series" and the value of treatment were referred to by several speakers in the discussion that followed on "The Psychiatrists' Contribution to Mental Health of Children."

Potentialities of the Subnormal

Dr. A. D. B. CLARKE'S account of new work on releasing potentialities in the subnormal fascinated the audience. "Imbeciles" should no longer be "written off"; many could be trained to become at least partly self-supporting. Though they were slow to learn, they *could* learn—as had now been shown in the laboratory and confirmed in industrial workshops. They responded to ordinary incentives—rewards, praise, and self-competition—but they learnt much better from purposeful teaching than from the haphazard experiences of everyday life. To obtain the best results they must be taught when young—a lesson that should be passed on to all doctors and by them to mothers.

Dr. JOHN BOWLBY opened the afternoon session on "New Concepts" with a broad survey of the psychological (learning theory), medical (psycho-analytic), and ethological (zoological) frames of reference for theories of emotional and behaviour disorders. All three, he considered, were complementary and should be integrated. He discussed "species-specific behaviour patterns" and a theory of motivation derived from lower animals and extended to man. A delightful film followed, illustrating the behaviour of baby rhesus monkeys with hard or cuddly dummy mothers. Its main lesson, that "the mother should always be there," might well be applied to humans; but it served to illustrate how psychological and psychiatric problems are now being studied with the techniques of scientific medicine.

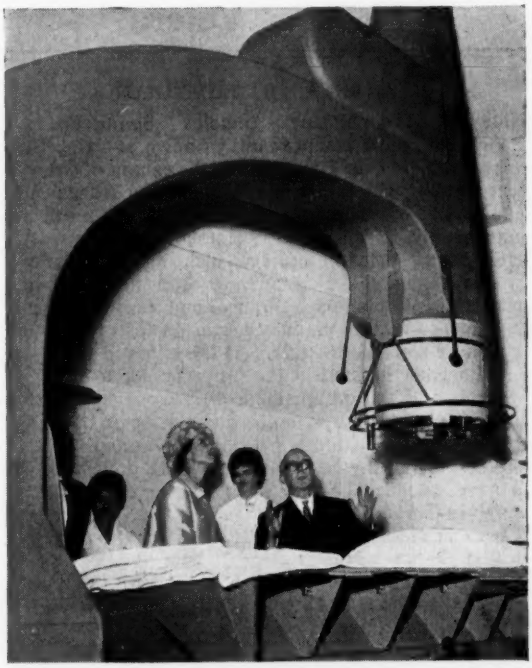
"Biochemistry and Mental Disorders" by Dr. GEORGE KOMROWER and "Genetics and Cytogenetics" by Mr. JOHN HAMERTON were so well summarized by the speakers that further condensation is not possible. With Professor PAUL POLANI ("gazing into the crystal ball") they shared the ability not only of advancing medical science but also of making recent discoveries clear to non-specialized medical men. If all their esoteric details were not completely shared with the audience, their scientifically controlled enthusiasm most certainly was.

A Whole Child

Dr. APLEY summed up what had obviously been a successful and enjoyable meeting. He urged further friendly co-operation between paediatricians and those of their colleagues who had had a formal training in psychiatry and psychology; the experiments described had proved of great value, directly for the patients and indirectly by their educative value to all those taking part. He looked on the paediatricians' growing interest in mental health as a non-competitive and natural expansion of their role as advisers in all aspects of the child's health, and he deprecated a too rigid separation of "organic" and "non-organic" factors. All of them there, he said, were trying to help a whole person—a child in the background of his family and environment: if they took the child apart to study him, they should remember to put the bits together again.

QUEEN VISITS GUY'S NEW SURGICAL BLOCK

H.M. QUEEN ELIZABETH, who is Patron of Guy's Hospital, visited New Guy's House on July 5 and unveiled a commemorative plaque recording her visit. The building, which accommodates 378 patients, has been in use for some months, and the Queen, who particularly wished to see the new block in its normal running order, visited wards, several of the departments, and toured one theatre suite.



The Queen inspects the linear accelerator in the radiotherapy department of New Guy's House.

£2½m. Building

The new eleven-story block has been built at a cost of nearly £2½m. on a seven-acre plot cleared by air raids, and it occupies the southern half of a site which can be further developed to the east and north. The radiotherapy department is on the lower ground floor, where are a linear accelerator, two caesium units, deep and superficial *x*-ray units, the Central Sterile Supply Department—this will eventually supply the whole hospital, with the exception of theatre instruments, which will be sterilized on the theatre floor — staff changing-rooms, and the air-conditioning machinery. The casualty department occupies the whole of the ground floor. The first floor houses the eight main operating theatres, arranged in four pairs. One of each pair has an observation dome which is reached from the floor above. Though the units are basically identical, each pair has some modification designed to cater for a specialist branch of surgery in addition to general surgery, theatre suite No. 1 being specially designed for thoracic surgery, No. 2 for E.N.T. work, No. 3 for orthopaedics, and No. 4 for urology. The ancillary services include a recovery ward, central instrument service, and a clinical lecture theatre which will hold 50 people. On the second floor are the departments of surgery, gynaecology, obstetrics, anaesthetics, and clinical microscopy, with research rooms and laboratories. Common to all departments are a

library, exhibition rooms, and surgeons' lounge. On the second floor are the four observation domes for viewing operations in the theatre floor below.

Traditional Wards

The wards are on the third to the ninth floor. Each floor is planned in the same way, and includes a central concourse, a number of service rooms, and two L-shaped ward units. Each unit has 27 beds, 12 in each wing of the open ward, and three beds in single rooms.

(Architect: Mr. A. S. Gray, of Messrs. W. H. Watkins, Gray, and Partners; consulting architect: Mr. Paul G. Burt, Chicago, U.S.A.)

Nova et Vetera

ICONOCLASTIC PHYSICIAN

Philippus Theophrastus Aureolus Bombastus ab Hohenheim, more compendiously known as Paracelsus, was born in 1493 at Einsiedeln, in the canton of Schwyz near Zurich. His father was Wilhelm von Hohenheim, a student of Tübingen, who practised medicine, subsequently, at Villach in Carinthia. Paracelsus, who had been instructed in medicine, alchemy, and astrology by his father, as a wandering student visited the universities of Germany, France, and Italy. There is no evidence that he graduated as a doctor of medicine though he assumed the title. At the mines in the Tyrol and Bohemia he studied the properties of metals and minerals and the occupational diseases of the miners, being thus enabled to write the first treatise on industrial diseases. He acquired a patron in Sigismund Fugger, one of the wealthy banker princes. He professed also to have extended his travels in pursuit of knowledge as far as Turkey, Egypt, and Tartary.

Back in Germany, by his disregard for the approved medical therapy of the time and by unorthodox methods, Paracelsus became so renowned for his cures of eminent persons that in 1526 he was appointed town physician at Basle and lecturer on medicine at the university. His symbolic act of burning the works of Galen and Avicenna as an introduction to his lecture course typified his position as an independent investigator. This and his unorthodoxy incurred the hostility of the physicians, and their enmity was fostered by the vanity, aggressiveness, and intemperate habits of Paracelsus. Driven from Basle in 1528, he wandered over Europe, sometimes befriended by kings and princes, at other times in squalor and poverty, continually disputing, writing, experimenting, taking large fees from rich patients, and giving free treatment to the poor. In the end he turned to religion, styled himself "Doctor of Theology," became milder in character and speech, and died in the Hospital of Saint Sebastian at Salzburg in 1541.

Withal a genius, he was, as Singer said, a writer of excessive obscurity, of language and of form as well as thought; few can penetrate to his full meaning. As the author of this book* states, in reading Paracelsus we must understand the system of medicine and philosophy against which he rebelled. Moreover, Paracelsus had vision. He deflected alchemy into a direction which led on to chemistry. He applied his experiments and his experience to the healing of disease. His humane attitude towards mental disorders was far in advance of his time. His *Great Surgery Book* precedes Ambroise Paré in the recognition of cleanliness and the self-healing properties of wounds. He made new chemical compounds and introduced tincture of opium. He would receive greater credit as a medical pioneer if his writings had not been mixed with mysticism, thaumaturgy, alchemy, astrology, and other beliefs and superstitions of his age. Out of the twenty-four books ascribed to Paracelsus himself some painstaking research student may one day winnow the wheat from the chaff to assess the true value of his contributions to medicine. In the meantime, this scholarly and interesting biography gives a graphic picture of the iconoclastic physician and his times.

ARTHUR S. MACNALTY.

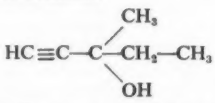
**Paracelsus. Magic into Science.* By Henry M. Pachter. (Pp. 360+x; illustrated. $4.) New York: Henry Schuman. 1961.

To-day's Drugs

Methylpentynol

"Atempol" (Norgine).
"Oblivon" (British Schering).
"Somnesin" (British Drug Houses).

Chemistry.—This is 3-methyl-1-pentyn-3-ol and has the following structural formula:



Pharmacology.—The drug is an orally active depressant of the central nervous system used chiefly for its calming, sedative, and hypnotic actions. It is rapidly absorbed, effects coming on in from 15 to 30 minutes and lasting one hour or more. The distribution in the body is similar to that of ethyl alcohol; it enters most cells and also crosses the blood–brain barrier and the placenta. Small amounts can be found in the saliva, milk, gastric secretion, aqueous and vitreous humour, amniotic fluid, bile, and urine. It can also be detected in the expired air. In man,[1] methylpentynol and its carbamate persist for 48 hours after withdrawal, and daily doses lead to rising blood concentrations.

Toxicity tests on various animals have shown methylpentynol to be remarkably non-toxic; in doses much higher than those used therapeutically in man respiration is not depressed, and there are no effects on the heart or blood-pressure. Experimental overdosage in animals causes coma and eventually death from respiratory failure. Studies in cats and dogs suggest that its calming effect may be due to a depressant action on the reactivity of the reticular activator system and the thalamocortical circuits, and an anticonvulsant action, which it also possesses, due to a lowering of cortical excitability and blocking of cortical and inter-hemispheric processes of synaptic transmission.[2] This anticonvulsant activity is marked in laboratory animals, although it is not an effect which is made use of clinically.

Therapy.—Methylpentynol was originally introduced as a short-acting hypnotic with no hang-over effect. It has about 40% of the hypnotic activity of phenobarbitone and pentobarbitone,[3] and is useful as an occasional hypnotic in mild cases of insomnia, particularly when this is caused by nervous tension or apprehension. The dose for adults for this purpose is 500–1,000 mg., while for children aged from 1 to 10 years the dose varies from 250–500 mg. The drug is best given when the patient is actually in bed. This drug is no good as a substitute hypnotic for patients who have been taking heavy doses of barbiturates, but is sometimes useful as an occasional mild hypnotic for patients who cannot take barbiturates because of idiosyncratic reactions to them.

As a routine daytime sedative methylpentynol is not very useful because too high a percentage of the patients tend to become too sleepy,[4] and also because repeated daily use of this drug can easily lead to toxic effects owing to its tendency to build up in the body even with therapeutic doses. As a pre-anaesthetic medication methylpentynol is

especially useful for children and has been used most successfully in those undergoing tonsillectomy.[5] It does not interfere with the anaesthetic to be administered and recovery from the anaesthetic is not prolonged. It does not depress the respiratory or cough centres. The doses usually used for children under 8 years are 500–750 mg. and for those over 8 years 750–1,000 mg. The time of administration is important, and it should be 1½–2 hours pre-operatively.[6]

In obstetrics methylpentynol has been found helpful. One thousand mg. is given at the onset of labour, followed if necessary by 500 mg. hourly or at longer intervals. The usual total dosage used is 3,000 mg. Although this drug has no analgesic or antispasmodic activity it probably helps the mother to relax and is said to cause some degree of amnesia. It has no adverse effects on the foetus and can only be detected in cord blood in minute amounts.[7] The drug has also been given per rectum,[8] 3–4 capsules half an hour before the customary preliminary enema, and this dose repeated after three hours if necessary.

Although methylpentynol in small doses is said not to impair physical or intellectual activity, it is obvious that the use of this drug before such events as examinations and driving tests is most unwise.

Side-effects.—In therapeutic dosage methylpentynol seldom gives rise to side-effects, though it sometimes causes belching with unpleasant after-taste. It appears to potentiate the depressant actions of barbiturates and alcohol on the central nervous system. In car drivers small doses may give rise to over-confidence, leading to errors of judgment. In patients cured of chronic alcoholism this drug sometimes results in a reversion to the craving for alcohol.[9]

Methylpentynol has been given daily by mouth to human subjects for up to four-month periods in doses ranging from 100–400 mg. without producing any toxic effects.[10] A potential hepatotoxic action was suggested by the development of strongly positive cephalin flocculation tests during the use of this drug[11]; however, other studies[10 12] have not disclosed such changes. Exfoliative dermatitis developed in one patient who had received 1.75 g. methylpentynol.[13]

While symptoms of overdosage may follow the swallowing of a large amount (for example, a few grammes) at once or over a period of a few hours, it is important to remember that if the drug is taken regularly every day in therapeutic doses, especially the higher ones (for example, about 1 g.), overdosage effects may occur after a few days because of its cumulative action.[1] With overdosage the patient may experience dizziness and light-headedness. Mood deviations ranging from elation and euphoria, irritability, anxiety, and tension through to apathy and depression may also occur. Other toxic effects are loss of facial tone, dysarthria, tongue tremor, ptosis, diplopia, nystagmus, pupillary abnormalities, and ataxia. Dysphasia, paraphasia, distorted time perception, illusions, hallucinations, and impairment of memory for the toxic episode have also been reported.[14] The occurrence of a withdrawal syndrome has also been recorded.[14] Lethal overdosage produces coma with death due to respiratory failure. Death has occurred following a dose of from 4.5–6 g.,[15] but survival has followed the ingestion of 10 g.,[16] and also 6.25 g.[7] and 8.75 g. combined with 400 mg. pentobarbitone.[17]

Treatment of cases of overdosage consists in gastric lavage, artificial respiration with oxygen if necessary, and the use of pressor agents such as noradrenaline and methylamphetamine.[18] The routine care of a comatose patient should be instituted, and this should include anti-infective therapy.

N.H.S. Basic Price.—100 250-mg. capsules, 15s. 6d.

REFERENCES

1 *Clin. Sci.*, 1958, 17, 629.
2 *J. Physiol. (Paris)*, 1957, 49, 1.
3 *Science*, 1951, 114, 184.
4 *Int. Rec. Med.*, 1952, 165, 199.
5 *Brit. med. J.*, 1960, 1, 1560.
6 *Med. J. Aust.*, 1959, 2, 829.
7 *Lancet*, 1954, 2, 522.
8 Ibid., 1958, 1, 743.
9 *Brit. med. J.*, 1955, 2, 1087.
10 *N.Y. St. J. Med.*, 1952, 52, 572.
11 *Science*, 1952, 116, 663.
12 *Amer. Practit.*, 1952, 3, 23.
13 *Calif. Med.*, 1952, 77, 339.
14 *Brit. med. J.*, 1956, 2, 1467.
15 *Amer. J. clin. Path.*, 1953, 23, 129.
16 *Brit. med. J.*, 1955, 1, 268.
17 *Northw. Med. (Seattle)*, 1952, 51, 778.
18 *Brit. med. J.*, 1955, 1, 268.

Correspondence

Because of heavy pressure on our space, correspondents are asked to keep their letters short.

Congenital Dislocation of the Hip

SIR,—In the present treatment of congenital dislocation of the hip there are two bogies, both due to misinterpretation of *x*-rays:

1.—*"Fragmentation" of the head of the femur,* an alarming term suggesting that the bone has been broken into pieces by violence; I have also known the changes shown in the *x*-rays to be called avascular necrosis. These terms are used to describe a purely temporary effect which occurs fairly often in cases treated by controlled movement. My notion is that the head should be moulded to fit the socket at the earliest possible moment, and that this can only be done by active use. The stresses on the head as its shape is changed may produce disturbances of ossification

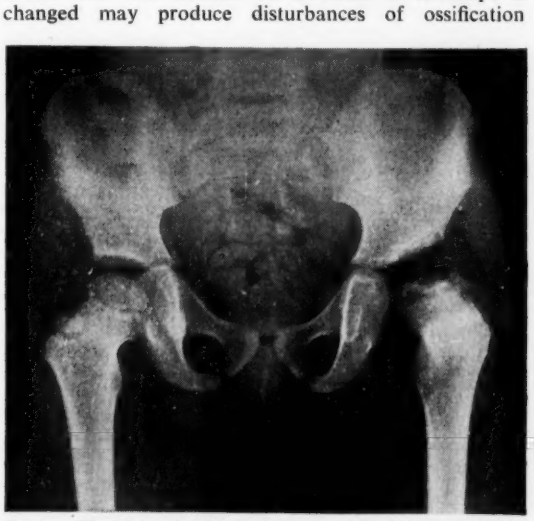


FIG. 1.—Hip-joints of child aged 3 years, taken 18 months after manipulation.

FIG. 2.—Hip-joints of same child at age 5 years.

FIG. 3.—Hip-joints of same child at age 10 years.

which have a vague resemblance to those of Perthes's disease, though there is an obvious difference from this in the apparently enormous joint-space, and clinically there is no pain or spasm.

From many similar cases I select the *x*-rays shown (Figs. 1–3): the child was immobilized for a month in plaster after manipulation, and then spent eight months of active locomotion in my splint. When the *x*-rays looked worst she was bouncing about and clinically normal.

2.—*Anteversion of the neck of the femur.* This is not of the slightest importance provided that the head is reduced into the acetabulum. The fashionable treatment by traction is very apt to leave it behind this socket, a displacement not visible on *x*-ray though detectable by the fingers. The resulting instability is often blamed on anteversion of the neck, the head twisted into the acetabulum at open operation, and the resulting rotation of the leg corrected by an osteotomy.

Two final remarks: (1) I think no one understands congenital dislocation of the hip till he has grasped why in its classical form it is common with valgus mouldings of the feet and unknown with varus ones.[1] (2) My main difficulty is to judge at what age manipulations should not be attempted.—I am, etc.,

London W.1. DENIS BROWNE.

REFERENCE

[1] Browne, D., *Aust. N.Z. J. Surg.*, 1959, **29**, 85.

SIR,—No one who is interested in congenital dislocation of the hip could fail to give full support to Mr. Norman Capener in his letter (July 1, p. 47), and no one could fail to praise the work done in Malmø by von Rosen in early diagnosis and treatment. But in quoting the case of Malmø, Mr. Capener has indicated the two main problems to be found in this connexion.

Firstly, Malmø is a very exceptional city. There is probably nothing quite like it in this country. The population is 250,000 and there is only one hospital, in which all the children are born. It is no great problem under these circumstances to organize that every baby is examined by a paediatrician and an orthopaedic surgeon within two or three days of birth. In this country the majority of babies are born at home, and those that are born in hospital are born in multiple hospitals, some large, many small, and often scattered. The problem of arranging overall competent diagnosis, such as obtains in Malmø, is obvious.

Secondly, the work of von Rosen in Malmø also suggests another aspect of the problem. During nine years, 39 diagnoses of congenital dislocation of the hip of all degrees have been made, all within the first few days of life. In the neighbouring city of Lund, Wiborg[1] has estimated that 22% of all osteoarthritic hips are due to congenital dysplasia of the acetabulum. As there is no reason to suppose that the incidence of osteoarthritis of the hip differs in Sweden from that in this country, the only possible conclusion must be that, even in the hands of such competent diagnosticians as are in Malmø, many minor degrees of dysplasia must be overlooked, but will turn up in 40 or 50 years as osteoarthritis. Such minor degrees of dysplasia are probably beyond the bounds of diagnosis and can only be dealt with by prevention, which we would suggest is quite different from the early diagnosis and treatment practised in Malmø. A condition which can be diagnosed has already developed and is beyond prevention.

There is evidence that in congenital dislocation of the hip the joint is primarily normal in shape and that displacement of varying degrees results from abnormal stretching of the capsule which at the time of birth may be pathological in that it will stretch, not like rubber, which is elastic, but like chewing-gum. Once stretched, it is in need of treatment, but until it has stretched it requires prevention only. At birth all hips have a flexion contracture, and extension, either passively or actively, will stretch the capsule, which if pathological may make displacement possible. If this capsular change is due to parental hormonal influence, which would be of a very temporary nature only, preventive measures need be of only short duration.

It seems increasingly probable that true prevention is possible and perhaps more practical than diagnosis in some areas. It is just a question of whether those responsible for delivery at birth can be persuaded of the importance of this, and if a means of preventing any extension of the hips from the moment of birth for a short period, perhaps only days, can be devised. We made many of these points in your correspondence columns last year (March 5, 1960, p. 724), but the subject is so important it needs repetition and emphasis.—We are, etc.,

E. W. SOMERVILLE.
J. C. SCOTT.

Nuffield Orthopaedic Centre,
Wingfield Morris Orthopaedic Hospital,
Oxford.

REFERENCE

[1] Wiborg, G., *Acta chir. scand.*, 1939, Suppl. 58.

SIR,—Your annotation (June 17, p. 1746) is a timely reminder that, as Mr. Norman Capener says (July 1, p. 47), "Congenital dislocation, as an established lesion, is now a preventable condition." The stress on early diagnosis is very correct indeed; on the other hand, there is nothing so unwilling as a congenital dislocation of the hip to disclose itself to those who are not looking for it, particularly before the child has walked. It is therefore essential, as Sir Denis Browne has pointed out, that newborn babies should each and every one be examined specifically to determine whether any hip abnormality is present or not. This may sound a monumental undertaking, but in fact it is not. Ortolani's test goes a long way towards solving the problem. It is, however, not infallible, and I have

devised a test which is more sensitive than Ortolani's and, so far, has picked out every hip abnormality in over 5,000 newborn children, proved by subsequent review at the age of 1 year.

The test consists of placing the child on its back with the legs pointing towards the examiner, with the hips flexed to 90° and the knees flexed. The middle finger of each hand is placed over the greater trochanter, and the thumb of each hand is placed on the inner side of the thigh approximately opposite the position of the lesser trochanter. With the thighs in mid-abduction, forward pressure behind the great trochanter is exerted by the middle finger of each hand in turn, the other hand holding the opposite femur and pelvis steady; if the femoral head is found to slip forward into the acetabulum, then the hip is dislocated. If there is no movement of the femoral head, then the hip is not dislocated. That is the first part of the test. The second part of the test consists in pressing downwards and backwards with the thumb on the inner side of the thigh. In a few hips, although the femoral head is in the acetabulum, such downward and backward pressure can cause it to slip on to the posterior lip or even further backwards. The femoral head slips back again into the acetabulum immediately the pressure is released. Such hips are described as "unstable"; they are not dislocated, but they are dislocatable. This test is, in my experience, very reliable, and can be used up to the age of 6 months, after which the child's femur has become longer than my hand, and I cannot reach the trochanter with my finger-tips.

This test reveals that hip laxity even to the point of dislocation is far more common than was previously thought, and that 88% of these hips undergo a spontaneous recovery. The test is simple to carry out, takes five seconds, can be taught to midwives, and it is a simple matter to arrange that any abnormal hip is referred immediately to a centre where management of this condition can be undertaken.—I am, etc.,

Salford, Lancs. T. G. BARLOW.

Abdominal Wound Dehiscence

SIR,—The recent correspondence about "burst abdomens" (March 25, p. 899) has drawn attention to this serious complication, and the incidence would appear to be increasing. In one hospital during a period of three months 11 abdominal wounds dehisced (see Table). Two of the cases were sutured with through-and-through black silk and the remainder with Ethicon catgut. Eight of these ruptures occurred less than 12 days after the original operation. Of these eight, four had some infection in the wound. On one surgical unit during the period January to April 67 upper abdominal incisions were sutured with Ethicon catgut and eight of them dehisced. These 67 cases included 20 gastrectomies and 14 cholecystectomies. The brand of catgut was then changed to London Hospital and in the ensuing ten weeks only one dehisced wound has occurred. At resuture of this last case on the tenth day the catgut was intact, but had been torn out of the peritoneum over a fairly wide area, and out of a smaller area in the rectus sheath.

While it is fair to say that the infection-rate in these wounds was high, that some of the patients were elderly, and that two had carcinoma, the cause of the dehiscence was considered to be fraying of the catgut and too early absorption.—I am, etc.,

London W.1. H. A. KIDD.

Panic Immunization

SIR,—No one will disagree with Dr. Guy Bousfield (July 1, p. 43) in his desire to avoid hasty and slip-shod immunization, but it would be most unfortunate if some of his statements went unchallenged, and, as a result of this, a considerable number of children were to remain unprotected against diseases which still kill and are not yet eradicated from the population.

I refer, in particular, to his allusion to the dangers of inoculating "allergic" subjects—a danger which has been greatly over-exaggerated and which is considerably less than the chance of a child being killed or injured through riding in a vehicle to the place of inoculation. It is not generally appreciated that *less than half* the reported deaths from anaphylactoid shock occur in so-called "allergic" subjects, and such deaths may occur at any age in persons who have never exhibited any form of allergic reaction previously, and in whose family there has been no such occurrence. This does not mean that there is not an increased risk in persons who suffer from, or have suffered from, one of the atopic disorders, but that the risk, even in this group, is extremely slight; *far more important* is the history of a previous reaction to the substance about to be injected or a close congener of it.

I do not consider it is ever justifiable to recommend that a child be refused protection against poliomyelitis, diphtheria, or tetanus merely because it happens to have eczema or another atopic disorder. As long as the risk of acquiring these diseases exists *all* children should be protected as soon as possible. Smallpox vaccination in

Abdominal Wound Dehiscence

Patient			Diagnosis	Date of Operation	Date of Burst	No. of Days Post-op.	Surgeon	Bacteriology
Sex	Age	Type of Admission						
F	70	Waiting list	Recurrent cholecystitis; cholecystectomy	8/12/60	19/12/60	10	A	11/12/60 *Staph. aureus* and *Proteus*. (Wound)
F	55	,, ,,	,, ,, ,, and appendicectomy	10/1/61	14/1/61	4	B	No infection reported
M	79	Emergency	Melaena from anastomotic ulcer; Roux-en-Y anastomosis	28/12/60	14/1/61	17	A	6/1/61 *Proteus morgani*, *Staph. aureus*. Ditto 12/1/61. (Wound)
M	62	,,	Acute intestinal obstruction. Adhesions divided	31/1/61	3/2/61	3	B	No infection
M	63	,,	Acute cholecystitis. Cholecystectomy	2/2/61	11/2/61	9	B	4/2/61 *Staph. aureus*. (Wound)
F	69	Waiting list	Carcinoma pancreas. Choledochoduodenostomy	4/2/61	17/2/61	12	C	19/2/61 ,, ,, ,,
M	46	,, ,,	Duodenal ulcer. Partial gastrectomy	10/3/61	13/3/61	3	A	11/3/61. Sputum—*Strep. pneumoniae*, and *Haemophilus influenzae*
M	64	Emergency	Haematemesis—D.U. Partial gastrectomy	10/3/61	21/3/61	11	C	No infection
M	72	Waiting list	Carcinoma middle third oesophagus. Upper abdominal laparotomy, thoracotomy, and oesophagectomy	5/3/61	13/3/61	8	D	,, ,,
N.B. Died 3 days after re-suture								
F	54	Emergency	Acute intestinal obst. Adhesions following oophorectomy 8/1/60 for Ca ovary	4/1/61	24/1/61	19	A	*Staph. aureus* in chest and wound
				N.B. Abdomen closed with through-and-through black silk				
F	38	,,	Pyosalpinx. Bilateral salpingectomy	2/3/61	16/3/61	14	A	Wound infection and breakdown to sheath only. Swab 3/3/61 *Escherichia coli*.

the atopic infant or child is a different matter, since there is a very real risk of the child developing a widespread eczema vaccinatum which carries with it a certain mortality, but in areas where variola major is endemic this is a risk which may have to be taken.

I am sorry that Dr. Bousfield feels it necessary to resort to the same stratagem as that of which he accuses his opponents, and thus convey a distorted picture of the true position regarding inoculation of persons with atopic disorders.—I am, etc.,

Guy's Hospital,
London S.E.1. E. J. MOYNAHAN.

SIR,—Dr. Guy Bousfield's excellent article on "Panic Immunization" (July 1, p. 43) comes as a timely reminder on the subject of allergy with respect to immunization procedures. There seems to be evidence of an absolute as well as relative increase in allergic phenomena in the child population of this country. With the now extensive use of triple antigens time should always be allowed for a full inquiry into family history of allergy, constitutional peculiarities, and previous severe reactions.

Perhaps the number of immunizations per session he suggests may be too many to maintain a safe, civilized, and humane procedure. Would not these procedures best be carried out in the surgery at a small special session by the child's own doctor, who may be well acquainted with the family history?—I am, etc.,

Hanworth, Middlesex. A. R. BROADBENT.

Hypotensive Drugs in a Heat Wave

SIR,—A small point but of practical importance: during a heat wave the response to ganglion-blocking and adrenergic-blocking drugs is enhanced, and orthostatic and exertional hypotension with fainting may become suddenly troublesome. The individual response to environmental rises in temperature varies, but it seems wise to be ready to diminish the dose of the drugs and to warn patients who may be going abroad on holiday of the need of reducing the dosage should symptoms become troublesome.—We are, etc.,

Hypertensive Clinic,
Whittington Hospital,
London N.19. E. MONTUSCHI.
P. PICKENS.

Underwater Swimming

SIR,—With reference to the annotation (June 3, p. 1596) on the risks of hyperventilation and underwater swimming, normally an underwater swim is terminated by an irresistible urge to inspire, which in Craig's cases[1] was apparently absent or overcome, resulting in loss of consciousness before drowning.

On two occasions I have had a similar experience, the latter leaving a vivid memory. After swimming about 30 metres I realized I had no desire to breathe and felt I could continue for ever. My mind seemed separate from my body, which appeared to swim automatically. I was able to reason that to continue must be dangerous, and so surfaced at 40 metres, taking a few shallow breaths only, in contrast to the usual gulp of air. For the next four hours I noticed a dull headache. Contrary to the other cases this was without hyperventilation, invalidating the proposed hypothesis in part.

In my experience the conditions decreasing the desire to breathe underwater are: (1) Water temperature above 65° F. (18.3° C.). (2) Moderate rather than deep inspiration on diving. (3) Initial pulse rate below 65 per minute. (4) Relaxation and swimming with the minimum effort. (5) Training. (6) Hyperventilation which I do not practise because it leaves me with a headache. I am not certain that depth has any effect; and confirm that swallowing temporarily relieves the urge to inspire and that the longest distances are swum in competition or with a set goal.

Besides saving lives of swimmers, research into this problem might reveal the physiology of a baby's first breath, to which the conditions are similar.—I am, etc.,

The Middlesex Hospital,
London W.1. B. M. MALE.

REFERENCE

[1] Craig, A. B., *J. Amer. med. Ass.*, 1961, **176**, 255.

Diagnosis of Tuberculous Meningitis

SIR,—I am writing with respect to your leading article (May 6, p. 1305) concerning the difficulty of making a diagnosis of tuberculous meningitis. I was surprised to find that no mention was made concerning the significant changes in fluid and electrolyte metabolism or the changes in permeability of the blood–brain barrier. Such changes are of help in establishing a correct diagnosis. Within the last month my own research laboratory has been called upon three times by attending paediatricians to investigate three patients with lymphocytic meningitis. One out of the three patients gave a positive result for tuberculous meningitis before the diagnosis could be established by other means. Brief discussion of these changes in electrolyte metabolism would seem justifiable at this juncture.

Ten years ago Rapoport, West, and Brodsky[1] drew attention to the inability of three patients with tuberculous meningitis to conserve or clear chloride loads and classed the disturbance in the group of salt-losing conditions. Their patients had hyponatraemia, hypochloraemia, and a raised plasma carbon-dioxide tension with plasma *p*H values on the alkaline side. Harrison, Finberg, and Fleishman in 1952[2] pointed further to the loss of potassium from muscle cells but did not study acid-base balance. Cheek in 1954[3] and again in 1956[4] drew attention to the presence of hypotonic expansion of extracellular fluid in tuberculous meningitis without significant loss of body chloride on a body-weight basis, so that such patients do not suffer from the low-salt syndrome. This conclusion is further supported by the deleterious effects of giving 3% saline.[5] The hyponatraemia in our studies was associated frequently with metabolic alkalosis—an acid-base imbalance that does not occur in other forms of lymphocytic meningitis, in lead poisoning, or in cerebral tumour in my experience. Admittedly from the study of some 30 patients with tuberculous meningitis (published and unpublished) it became apparent that the more advanced the disease the worse the changes in plasma electrolyte concentration. Also these changes are independent of overt fluid losses and are probably related to the metabolism of the tuberculous organism since they occur also in miliary tuberculosis. However, even at the onset of tuberculous meningitis there is alteration of the permeability of the blood–brain barrier, which can be shown by the diffusion of univalent ions such as bromide. This was first shown by Walter in 1929,[6] by Taylor, Smith, and Hunter in 1954,[7] by Cheek in 1956,[4], by Nicol and Fawns in 1958,[8] and by Crook *et al.* in 1960.[9] Indeed Nicol and Fawns, in a series of 26 cases of early undiagnosed lymphocytic

meningitis, showed that every case subsequently confirmed as tuberculous meningitis also had a $\frac{\text{plasma Br}}{\text{C.S.F. Br}}$ ratio of less than 1.4 (normal 3.0)

Our own approach to this problem has been to give suspected children 2 ml./kg. of 2% sodium bromide orally, intravenously, or subcutaneously to measure the corrected bromide space and the $\frac{\text{plasma}}{\text{C.S.F.}}$ bromide ratio 12–24 hours later, as well as serum electrolyte concentrations. Normal values in children for the corrected bromide space, which closely measures extracellular volume, have been reviewed recently,[10] also methods of serum bromide determination. The fall in C.S.F. chloride concentration would seem to depend on the usual dilution of the extracellular fluid phase in this disease and the impairment of the blood–brain barrier so that the C.S.F. chloride gradually equilibrates with the low plasma chloride concentration.—I am, etc.,

Research Foundation,
Royal Children's Hospital,
Melbourne, Australia. DONALD CHEEK.

REFERENCES

1 Rapoport, S., West, C. D., and Brodsky, W. A., *J. Lab. clin. Med.*, 1951, **37**, 550.
2 Harrison, H. E., Finberg, L., and Fleishman, E., *J. clin. Invest.*, 1952, **31**, 300.
3 Cheek, D. B., *Pediatrics*, 1954, **14**, 87.
4 ——ibid., 1956, **18**, 218.
5 Doxiadis, S. A., Goldfinch, M. K., and Philpott, M. G., *Brit. med. J.*, 1954, **1**, 1406.
6 Walter, F. K., *Die Blut-Liquorschranke*, 1929. Thieme, Leipzig.
7 Taylor, L. M., Smith, H. V., and Hunter, G., *Lancet*, 1954, **1**, 700.
8 Nicol, V. S., and Fawns, H. T., *Arch. Dis. Childh.*, 1958, **33**, 440.
9 Crook, A., Duncan, H., Gutteridge, B., and Pallis, C., *Brit. med. J.*, 1960, **1**, 704.
10 Cheek, D. B., *J. Pediat.*, 1961, **58**, 103.

Cortical Mastoidectomy

SIR,—I have read with interest and approval the paper by Mr. R. S. McAlary (June 24, p. 1802) indicating the need which still exists for the cortical mastoid operation in children suffering from acute suppurative otitis media which has persisted despite an intensive course of antibiotics. The cases which he has described show by the presence of granulation tissue in the cells that the infection had been checked by the antibiotics, but not destroyed. This is the danger which we must be aware of because such patients might develop a chronic suppurative otitis media and lose their hearing, especially now that most antibiotics are taken orally, often in inadequate dosage.

Thy operation is still needed for two other diseases whicc, are becoming more common. Firstly, the "gluey" ear which occurs in children whose presenting symptom is deafness. The drum looks dull and grey and audiogram shows a 20/30 decibel loss in the speech frequencies. These ears contain sticky mucus and when this has been removed after myringotomy the hearing improves, but often deteriorates again. A cortical mastoid operation would then be necessary in order to remove the thickened mucous membrane in the cells and prevent the deafness recurring. The other indication is the "blue" drum due to blood and pearls of cholesterol in the middle ear and mastoid air cells, described by Birrell,[1] associated with cholesterol granuloma. These patients are deaf, but when the blood, etc., has been removed from the middle ear and mastoid the hearing improved.

I hope that Mr. McAlary will not mind my disagreeing with him on the indications for this operation, which are no longer accepted by most otologists. A facial palsy occurring during an acute otitis media usually improves with the treatment given for the infection, and otitic meningitis usually complicates chronic suppurative otitis media, but when it occurs in the acute variety the meningitis must be treated first.—I am, etc.,

E.N.T. Infirmary,
Liverpool 1. J. SIEGLER.

REFERENCE

1 Birrell, J. F., *J. Laryng.*, 1958, **72**, 769.

Medical and Surgical Vagotomy

SIR,—We were most interested in the comments of Mr. P. G. Bevan, Mr. B. N. Brooke, and Mr. G. Slaney (June 24, p. 1827) and of Mr. G. E. Moloney (p. 1828) relating to our paper "The Effect of Medical and Surgical Vagotomy on the Augmented Histamine Test in Man." We appreciate their provocative discussion and wish to reply to one or two points raised by them.

Mr. Bevan and his colleagues rightly indicate that the absolute level of post-operative gastric acidity may bear no correlation with recurrent ulceration. It was for this reason, and because there is such a wide variation in the absolute acid outputs in any series of duodenal ulcers, that we studied the proportion by which the gastric acid is reduced in the individual case and reported percentage reductions following either medical or surgical vagotomy. Mr. Bevan refers to their results reported in the *Lancet*[1]; in these studies, arbitrary sub-maximal doses of histamine and of other conventional stimuli were used (as opposed to our maximal histamine stimulation) and acid measurements were not presented.

No one would deny that there are inevitable difficulties in comparing the results of gastric-secretion tests after a gastro-jejunal anastomosis with the results of similar tests before the operation; this is largely due to the possibility of loss of acid through the stoma and reflux of alkaline duodenal juice into the stomach. Far from evading this issue, we discussed it in some detail and referred to previous observations of our own in which a statistical study of the effect of gastro-jejunostomy alone was made.[2] Furthermore, the observations of Kyle and Stephens,[3] who performed augmented histamine tests after vagotomy with gastro-jejunostomy and during these tests blocked the stoma by means of a balloon, show that the slight increase in the recovery of gastric juice was not of sufficient degree to justify the routine use of this difficult and tedious technique in clinical studies. These workers claim that any inaccuracy due to loss of gastric juice or of reflux of duodenal content is unlikely to effect significantly conclusions based on post-operative acid-secretion studies. The surgical treatment of duodenal ulcer is aimed at the reduction of gastric acid secretion; the inevitable difficulties placed in our way by the necessity of providing a drainage procedure should not deter us from attempting to assess the physiological effects of these operations.

Mr. Moloney is to be congratulated on the excellence of the results he has obtained in a personal series of almost 250 cases of antrectomy with vagotomy for duodenal ulcer. From a search of recent reports on the use of this operation in clinics specializing in gastro-enterology, we have found that the overall immediate mortality is of the order of 2.5%; this compares

unfavourably with the figure or about 0.5% from vagotomy with a drainage procedure, and we still consider that the addition of gastric resection to vagotomy increases the risk to the patient. For this reason, we would not advise its routine use in the surgical treatment of chronic duodenal ulcer.—We are, etc.,

University Department of Surgery, The Royal Infirmary, Sheffield 6.

I. E. GILLESPIE.
A. W. KAY.

REFERENCES

1 Slaney, G., Bevan, P. G., and Brooke, B. N., *Lancet*, 1956, **2**, 221.
2 Tankel, H. I., Gillespie, I. E., Clark, D. H., Kay, A. W., and McArthur, J., *Gut*, 1960, **1**, 223.
3 Kyle, J., and Stephens, F., *Brit. J. Surg.*, 1961, **48**, 465.

Primaquine Sensitivity

SIR,—We read with interest the article entitled "Primaquine-Sensitivity of Red Cells in Various Races in Southern Africa," by Drs. R. W. Charlton and T. H. Bothwell (April 1, p. 941). In their series, 3% of Bantu males have shown an unstable pattern of reduced glutathione with low glucose-6-phosphate dehydrogenase activity in red cells, while in 100 Indian males from Natal no such abnormality has been detected.

In this connexion, our findings on Indians may interest the readers. In approximately 5% of normal Indians, predominantly Bengalese, the reduced glutathione in red cells showed a characteristic unstable pattern in association with a reduced level of glucose-6-phosphate dehydrogenase activity.[1 2 3] In a series of six cases of haemoglobinuria investigated by us during the last 18 months, typical pattern of reduced glutathione instability was detected in as many as five. "The Indians in Natal were mostly descendants of immigrants from Madras." This difference in the enzymatic patterns may, however, be explained by assuming that gene frequency of this disorder varies in different parts of India, as discussed by Drs. Charlton and Bothwell. Such differences in gene frequencies are also noticeable with reference to abnormal haemoglobins as reported from different parts of India.[4]—We are, etc.,

J. B. CHATTERJEA.
S. SWARUP.
S. K. GOSH.

Haematological Unit, Indian Council of Medical Research, and Department of Haematology, School of Tropical Medicine, Calcutta, India.

REFERENCES

1 Swarup, S., Ghosh, S. K., and Chatterjea, J. B., *Nature (Lond.)*, 1960, **188**, 153.
2 —— —— *Bull. Calcutta Sch. trop. Med.*, 1961, **9**, 8.
3 —— —— *Indian J. Path. Bact.*, 1961, **4**, 6.
4 Chatterjea, J. B., in *Abnormal Haemoglobins: A Symposium Organized by the Council for International Organizations of Medical Sciences*, 1959, p. 327, edited by J. H. P. Jonxis and J. F. Delafresnaye. Blackwell Scientific Publications, Oxford.

Phenindione and Blood-coagulation Factors

SIR,—In reply to Professor P. A. Owren's letter of June 10 (p. 1679), I would like to clarify two points.

(1) My Christmas-factor assay method employing Prower–Stuart-factor eluate was not intended to give accurate absolute values, but was applied because of the availability of Prower–Stuart eluate as a means of following day-to-day variations of Christmas-factor levels in the presence of low values of Prower–Stuart factor during therapy. The method may well give slightly high values. It was felt, however, that it would differentiate between the zero values reported by Johnson *et al.*[1] and the much higher values obtained by other workers.

(2) I do not question the usefulness of the thrombotest reagent in the control of anticoagulant therapy, but rather the *degree* of sensitivity to Christmas factor at therapeutic levels of all factors. This was the hazard to which I referred. In applying the simple test which Professor Owren mentions, I am reminded that when a Christmas disease plasma having 1% Christmas factor is diluted 1 in 10 (giving a final Christmas-factor concentration of 0.1%) and other factors are maintained at the 10% level, the ratio of Christmas factor to factors VII and X is 1 to 100. It is a big jump from haemorrhagic levels of 10% to levels of 0.1%. The pertinent question is, "What is the sensitivity of the thrombotest reagent to haemorrhagic Christmas-factor levels of 5% to 10%? If in this simple test the two diluted plasmas are mixed in equal proportions, thus giving a mixture containing 5% Christmas factor and 10% of other factors, the prolongation in clotting time of the mixture beyond that of the 10% normal is three to four seconds. In the thrombotest correlation curve, the difference in clotting times between 10% and 5% dilutions of normal plasma is over 80 seconds, and the clotting time of a plasma containing 0.1% of Prower–Stuart factor is measured in minutes and not seconds. Thus a small rise in Prower–Stuart factor may mask a much greater coincident reduction in Christmas factor, resulting in a shorter clotting time (or higher percentage) by the thrombotest method, when in fact the Christmas factor level is falling rapidly. Experimental evidence was provided in my paper to demonstrate this.

The thrombotest reagent does have a definite slight sensitivity to Christmas factor, and I would join Professor Owren in welcoming any reagent which may herald a risk of bleeding complications in patients under anticoagulant therapy. I simply exercise caution in the interpretation of the word sensitivity. I have had experience with the thrombotest reagent for 18 months and would have no hesitation in recommending it to the small laboratory or the larger laboratory desirous of saving the time of technicians in preparation, standardization, and manipulative procedures involved in conventional methods.—I am, etc.,

KENNETH W. E. DENSON.

St. Pancras Hospital, London, N.W.1.

REFERENCE

1 Johnson, S. A., Priest, E. M., and Caldwell, M. J., *Angiology*, 1959, **10**, 435.

SIR,—Professor P. A. Owren (June 10, p. 1679) in commenting on Mr. K. W. Denson's article on "Phenindione and Blood-coagulation Factors" (April 29, p. 1205), made a number of statements concerning Quick's prothrombin time which deserve critical scrutiny and correction. He emphasizes that bleeding occasionally occurs when "Quick's prothrombin time is within the accepted therapeutic range." That statement is correct. When the prothrombin activity is reduced to what may be regarded as the protective level against thrombo-embolism, a potential bleeding state is produced. This is a hazard that has to be taken, but such haemorrhages are not nearly as serious as a recurrent thrombo-embolic attack.

Professor Owren's second point—namely, "The one-stage test of Quick gives a measure of the combined effect of prothrombin, factor VII, and factor X (Stuart–Prower factor)"—is not strictly correct. After the administration of a coumarin drug, factor VII decreases first and, later, prothrombin. The basic one-stage test measures only the fall of factor VII because the effect of the decrease of the two factors is not additive. By a simple additional step—adding a small quantity of aged serum—the one-stage test becomes specific for prothrombin.[1]

The third statement—"The haemorrhagic syndrome caused by overdosage of anticoagulants resembles that seen

in haemophilia and Christmas disease"—needs particular scrutiny because one can amplify it by adding that the bleeding also resembles that seen in hereditary hypoprothrombinaemia vera and severe hereditary factor-VII deficiency. In the latter two diseases, haematuria, haemarthroses, and deep intramuscular bleeding are observed.[2]

Much stress has been placed by Professor Owren on the depression of factor IX by coumarin drugs, and little mention is made of their effect on decreasing the prothrombin level. In hereditary hypoprothrombinaemia vera in which factors V, VII, VIII, IX, and X are normal, a mild bleeding tendency may appear in an individual whose prothrombin time is prolonged to only 17 seconds. This, when calculated on our prothrombin activity curve, is 40% of normal. It has been found in our laboratory that, when the basic prothrombin time is maintained at 25 seconds in patients on a long-term anticoagulant regime, factor VII is approximately 20% of normal, while prothrombin is usually at a 30–40% level.

In agreement with Mr. Denson and with Zollinger,[3] we have not found the thrombotest sensitive to factor IX. When the test was carried out on the blood of two patients with severe haemophilia B (Christmas disease), the results were in the range obtained on normals and on classical haemophiliacs. It should be pointed out that factor IX is in an inactive state in circulating blood, and therefore the interpretation of the results obtained with tests of the partial thromboplastin type deserves great caution.—I am, etc.,

ARMAND J. QUICK.

Marquette University School of Medicine,
Milwaukee, Wisconsin, U.S.A.

REFERENCES

[1] Quick, A. J., *Hemorrhagic Diseases*, 1957, p. 382. Lea and Febiger, Philadelphia.
[2] —— Pisciotta, A. V., and Hussey, C. V., *A.M.A. Arch. intern. Med.*, 1955, **95**, 2.
[3] Zollinger, W., in *Thrombosis and Anticoagulant Therapy*, 1960, p. 42, edited by W. Walker. Thompson, Dundee.

Chloroquine and the Eye

SIR,—The ever-increasing use of chloroquine and its derivatives in this country for the treatment of rheumatoid arthritis, lupus erythematosus, and tapeworm, prompts me to write and add one more plea to those that have appeared elsewhere for caution in the use of this drug.

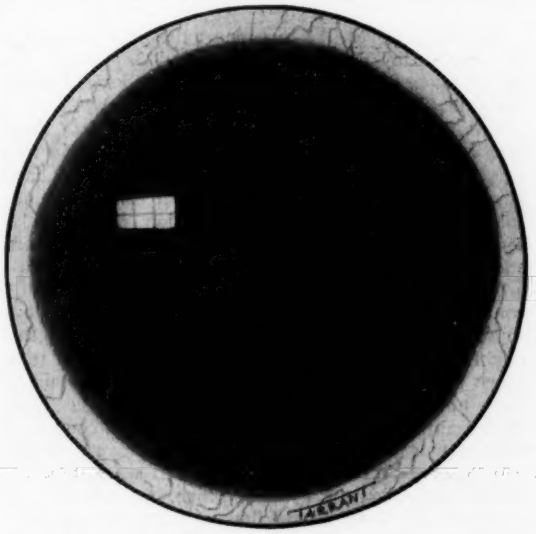
As has recently been shown by Hobbs, Eadie, and Somerville,[1] and by many previous workers, the effect of chloroquine on the eye is twofold—corneal and macular; and, although the former may be the more dramatic, the latter is frequently irreversible and productive of a permanent defect of vision.

A case has recently come to my notice which showed a typical corneal condition and which is, I think, worthy of record.

A woman of 55 had been treated by her practitioner for rheumatoid arthritis with "nivaquine" (chloroquine sulphate) in the normal dosage of 600 mg. daily. The treatment started in the middle of March, and was continued for ten weeks. At the end of two months she was on holiday in Cornwall, and because of a blurring of her vision visited an optician in Looe. It seems that the corneal picture was further clouded by conjunctivitis, but other members of the firm were called in, agreed that something unusual was afoot, and advised her to return to her doctor.

I saw her on May 19 and found the following: Right vision 6/12, left vision 6/18 not improvable further with glasses. Both corneae showed a milky haze without vascularization, which was first thought to be in the nature of corneal dystrophy. It was not possible to examine the fundi and media further, but the fact that the acuity was not unduly depressed made it reasonable to suppose that the macula was not involved in this case. The tension was normal and the eyes otherwise apparently healthy, and the great similarity of the corneal picture to those published made it reasonably certain that this was another case of chloroquine poisoning. Since the drug has been stopped the corneal haze is clearing, and when the painting (see Fig.) was made at the beginning of June it had assumed the second stage when fine radiating lines emerge from a focus just below the corneal centre.

Retinal changes in this condition are less frequent than corneal, but are the ones which bring the patient to the doctor and which are productive of visual damage which may well be permanent. Symptoms complained of are a clouding of vision and flashes of light, and



occasionally of night-blindness. This latter symptom ties up with a gross field contraction and a fundus picture not unlike that of retinitis pigmentosa. In addition the macular area may present a fine pigmentary stippling, with the visual acuity correspondingly reduced.

One can only hope that this and other warnings may induce in those who prescribe chloroquine a healthy respect for its toxic potentialities.—I am, etc.,

Worthing, Sussex. A. H. OSMOND.

REFERENCE

[1] Hobbs, H. E., Eadie, S. P., and Somerville, F., *Brit. J. Ophthal.*, 1961, **45**, 284.

Peak Expiratory Flow Rates

SIR,—Dr. John M. Jackson's thoughtful letter on this subject (June 17, p. 1760) will be read with sympathy. The men he has to assess as recruits to industry, while free from chest symptoms in the usual sense, are not as rigorously selected as those on which I based my figures (May 13, p. 1365). My purpose was to emphasize that, even in men who denied the occasional morning cough to which most smokers admit, the range of values is extremely wide. Apparently healthy men with minor respiratory symptoms found in most smokers were found to have significantly lower values, both by Mork,[1] who studied London Post Office workers, and by the College of General Practitioners.[2]

As Dr. Jackson remarks, the test can be biased by a number of factors, not least by lack of co-operation by

the subject. The prognostic significance of a low P.E.F. at ages under 40 is not known. Prospective studies are required to determine whether the decline in P.E.F. with age is uniform in all individuals, or whether some groups deteriorate more rapidly than others. Until factors which hasten the decline of P.E.F. with age are identified by such studies (one such factor might be heavy smoking), it would seem quite unjustifiable to exclude from any occupation a man who is capable of carrying out the work without distress.—I am, etc.,

Postgraduate Medical School, London W.12. C. M. TINKER.

REFERENCES

[1] Mork, T., Ph.D. thesis, University of London, 1960.
[2] College of General Practitioners, in preparation.

Connective Tissue

SIR,—We should like to support the general concept put forward by Dr. C. H. Lack (May 20, p. 1459) and by Drs. J. L. Potter and J. J. R. Duthie and Mr. George T. Watts (June 17, pp. 1758–1759).

Investigations recently completed here (Allan, Mitchell, and Hendry, in preparation) have shown that patients with intervertebral-disk derangements have evidence of a generalized upset in their ground-substance metabolism, with depression of the polysaccharide level of fascia as well as of nucleus pulposus, and an increase in serum protein-bound hexose, hexosamine, and glucuronic acid. An enzymatically controlled degradation of polysaccharide is the likeliest source of this abnormality, and confirmation of this might open the way to the specific treatment of a wide range of diseases. Owing to the difficulty of obtaining fresh normal human disks, we have for the last year been examining enzyme levels in other tissues concerned with polysaccharide metabolism: we hope very shortly to publish a preliminary report on this work, showing variations in enzyme activity which, we believe, add considerable weight to the primary "autolytic" pathology postulated by Dr. Lack, and which suggest, in accord with Drs. Potter and Duthie, that auto-immunity may be no more than a secondary phenomenon.—We are, etc.,

N. G. C. HENDRY.
A. J. CARR.
Royal Infirmary, Aberdeen.

Massive Paracentesis Abdominis

SIR,—Complications of rheumatic fever give rise to many problems. I should like to record the following case.

Mrs. H., of Italian parentage, was born here in 1916. Her previous history was scanty. It appears she had suffered from "joint pains" at the age of 16 years, for which she received "some medicine" but no bed rest. Following pregnancies in 1941 and 1945, against which she was advised (in view of a detected mitral murmur), she became a little dyspnoeic. She was put on to diuretics and digoxin and was able to carry out her household duties.

In January, 1949, she was referred for specialist opinion. At that time there were no congestive signs whatsoever. The heart was grossly enlarged. The signs were those of mitral stenosis and regurgitation, and probably tricuspid defect. *X*-ray findings confirmed this. Electrocardiogram showed gross sinus tachycardia with a tendency to right axis deviation. She refused hospital admission. In September, 1954, she agreed to go into hospital. On admission, she had had oedema of ankles for three days and increasing dyspnoea for three weeks. There were congested and pulsating neck veins, cyanosis of lips and nails, slight sacral oedema, and marked ascites. B.P. 140/90. Fibrillating pulse. Heart showed mitral stenosis and regurgitation, aortic stenosis and regurgitation, and tricuspid regurgitation. Liver enlarged, not tender. Spleen not felt. Considerable symptomatic improvement occurred after a month on strict bed rest, low salt diet, digoxin, and mersalyl.

In April, 1957, she entered hospital for paracentesis abdominis. Eight pints (4.5 litres) of fluid were withdrawn and she made satisfactory progress. Thereafter she entered hospital monthly. A total of 217 pints (127 litres) of fluid was withdrawn from April, 1957, to February, 1958. I first saw her in August, 1957. The mercurial diuretics were still having some effect. Liver still enlarged, heart sounds as before, spleen palpable three fingers' breadth below the left costal margin. In January, 1958, chlorothiazide produced a remarkable although temporary remission of her condition. In July, 1958, paracentesis was again undertaken. After July, 1959, I was prevailed upon to carry out this procedure in her home. By August, 1960, gross hypertrophy of the spleen was evident right down to the left iliac fossa.

A paracentesis abdominis can be readily undertaken in the home with the assistance of the district nurse and so release much-needed hospital beds. From the start of this procedure in April, 1957, until her death in May, 1961, a grand total of 932 pints (530 litres) of fluid was withdrawn from this lady's abdomen. I should be interested to learn if this is a record for such a short period.—I am, etc.,

Swansea, Glam. JOHN A. BOWLES.

Headache and "Eyestrain"

SIR,—Ophthalmologists are in confusion about their practice. May I offer a brief explanation? An error of refraction is constant. Headache is capricious, and is rarely the only symptom. It commonly arises at a time of emotional stress, and is grafted on to an underlying "neurotic" tendency, usually of the morbid anxiety type. In homely terms, heartache has been converted into headache; and glasses get the credit for what time, circumstance, and suggestibility cure. But we progress. Some forty years ago a leading ophthalmic surgeon claimed to have cured "shell-shock"—war neurosis— by correcting 0.125 D astigmatism. Will your correspondents join me in a sympathetic smile at the use of such potent magic?—I am, etc.,

Portsmouth. W. S. INMAN.

Cigarette in the Mouth

SIR,—My attention has been drawn to the letter from Dr. Geoffrey Barber (June 17, p. 1765), in which he suggests further inquiry into the lighting habits of smokers with bronchial carcinoma. Perhaps Dr. Barber may have overlooked the article by Professor A. Bradford Hill and Dr. R. Doll which you published in question-and-answer form,[1] especially the following:

Question 16.—Does the use of matches instead of a petrol lighter diminish the risk?

Answer.—This question was also studied in our inquiry into patients but has not, to our knowledge, been taken up elsewhere. We found no material difference in the use of petrol lighters between the cigarette-smoking patients with lung cancer and the cigarette-smoking patients with other disease: 42.9% of the former and 41.3% of the latter reported that at some period they had regularly used petrol lighters.

In 1957 a somewhat similar suggestion was made (not by a doctor), and on that occasion we immediately informed the Medical Research Council that we were ready and willing to give them any information or

assistance they might desire for the purpose of pursuing their inquiries. Our present attitude is, of course, the same, and we are anxious to be of assistance to any serious research worker.—I am, etc.,

G. P. BARTHOLOMEW,
Deputy Chairman,
Bryant & May Limited.

London E.3.

REFERENCE

[1] Bradford Hill, A., and Doll, R., *Brit. med. J.*, 1956, **1**, 1160.

Prevention of Warts

SIR,—I was delighted on returning from my Polish visit with the Dowling Club to read Dr. Neville Rowell's comments concerning prophylactic measures to prevent the spread of plantar warts (June 10, p. 1687). It seems incredible that in our day the practice of barefoot gymnastics and dancing should continue in spite of the overwhelming evidence that here is the major source of infection. In some quarters in Sussex the practice has become obsessional. It is a misuse of ratepayers' money to build elaborate schools for better education, and then have the children trooping up to hospital out-patients for treatment for their verrucae which could have been prevented by a little forethought.

The problem of verrucae in Poland was almost non-existent. It is not because there are no swimming-pools, for on the contrary Warsaw has one of the most up-to-date, and Cracow has two large open-air pools.—I am, etc.,

Hove 2, Sussex. E. COLIN-JONES.

Light After Dark

SIR,—I recently purchased a secondhand car of the £1,500 class which was fitted with all modern equipment, including non-essentials such as cigar-lighters and sliding ash-trays, but no electric socket in which to plug an inspection lamp for after-dark road repairs.

Electric torches are unreliable and candles blow out easily. The makers obviously have no imagination and cannot see beyond the end of their noses.—I am, etc.,

Leeds, Yorkshire. DAVID A. HERD.

POINTS FROM LETTERS

Chronic Headache

Mr. R. WALKINGSHAW, F.R.C.S.Ed. (Hobart, Tasmania), writes: Unfortunately no mention was made in your leading article (April 1, p. 957) of a condition which is very common here in Tasmania and elsewhere in Australia, and indeed, I am sure, is prevalent in Britain too when I recall some of the headaches I met when in practice there many years ago before I embarked on my service overseas. The headache to which I refer is easily recognized and was described in detail in a short paper which I presented to the Ophthalmological Society of Australia.[1] It always occurs in the forehead just above or behind the eyes, as indicated by the patient pointing in through the zygomatico-frontal suture. It comes on most days soon after breakfast, persists during the rest of the day, is described as a dull ache but is very persistent and so is very disabling, and is not much alleviated by aspirin and similar tablets. . . . The cause has been found to be deficiency of thiamine chloride brought on by a diet which is so predominantly carbohydrate as to be almost completely lacking in protein. Immediate relief is obtained by consuming a 10-mg. tablet of thiamine chloride with meals, but of course the real cure is to reconstruct the diet so as to include an adequate portion of protein at each meal.

REFERENCE

[1] Walkingshaw, R., *Trans. ophthal. Soc. Aust.*, 1959, **19**, 43.

Obituary

J. A. A. MEKELBURG, M.R.C.S., L.R.C.P.

Dr. Johannes August Albert Mekelburg, medical officer to Peek Frean and Co. Ltd., died suddenly on July 4 at London Airport after returning from a visit to Northern Ireland. He was 62 years of age.

John Mekelburg, as his colleagues always knew him, came from South Africa to England and qualified at Guy's Hospital in 1925. He worked in general practice, married his English wife, and settled in Forest Gate, where he continued to work until 1939. He then became medical officer to Messrs. Peek Frean and Co. in Bermondsey and established there a sound medical service, becoming a friend, colleague, and adviser to all employees, directors, managers, office, and work staff. He was a kind and very honest doctor, proud of his profession.

From 1939 onwards he devoted his whole time and energies to industrial medicine and was an early member of the Association of Industrial Medical Officers, becoming honorary secretary in 1949. With his energy, devotion, and enthusiasm, he was instrumental, with some colleagues of like mind, in building up the association until it has become a live professional body. Perhaps one of his greatest contributions, apart from his hard work as honorary secretary, was the work he did for the *Transactions*, the journal of the association, of which he was the honorary business manager from its inception in April, 1951, until his death. He saw its growth from a quarterly bulletin into a professional journal of good standing.

He was an energetic and devoted worker for his specialty, and in addition to his service to the Association of Industrial Medical Officers was a moving spirit in the Occupational Health Committee of the British Medical Association. He felt deeply on the use and place of medicine in industry and was notably interested in occupational skin disorders. His own success in dealing with the problems presenting themselves in the factories which he was looking after lent weight to his opinions expressed in committees and subcommittees. His interest in industrial problems in general was also shown in his active membership of the Royal Society for the Prevention of Accidents, and he thus helped his colleagues in industry to keep in constant touch with that admirable voluntary association.

His death is a personal loss to all his colleagues, and it comes as a particular shock to many of them because he seemed so well and to enjoy so much the recent admirably organized Annual Provincial Meeting of the Association of Industrial Medical Officers in Northern Ireland, which ended only a few days before his death.

Our deep sympathy goes out to his widow and children. In his family life John Mekelburg was a very happy man, and his wife and two daughters formed with him a devoted family group.—J. J. O'D.

ANIBAL RUIZ MORENO, M.D.

Professor Anibal Ruiz Moreno, professor of medical history in the medical faculty of the University of Buenos Aires and one of the leading rheumatologists in the Argentine, died on December 22, 1960.

Dr. MICHAEL KELLY writes from Melbourne, Australia: When, in a letter published in the *Journal* of April 29 (p. 1253), I mentioned the Argentinian scholar-rheumatologist Anibal Ruiz Moreno, I did not know that he had died suddenly at the end of last year at the early age of 56. He died while he was lecturing to students—as he would have wished, in the middle of the work he loved so well. For more than 20 years he had been director of the anti-rheumatic centre of the medical faculty of Buenos

Aires, and 24 years ago he founded the *Archivos Argentinos de Reumatologia*, which he edited until his death. He contributed a great number of articles to this and to other medical journals. He was president of the Argentinian Society of Rheumatology and for years he had been active on the Ligue Internationale contre le Rhumatisme: he had been a vice-president for eight years and was president-elect at the time of his death; he was to have been elected president at the Tenth Congress of the Ligue in September.

In addition, Ruiz Moreno was a great scholar and had been attached to the department of medical history in Buenos Aires for 25 years. In 1943 he was associate professor and ten years later he was appointed professor of the history of medicine. He wrote three books about South American medical history—one about mediaeval medicine, one on medicine in Greco-Roman mythology, and one each on the rheumatic diseases in the works of Hippocrates (1941), Sydenham (1943), Baillou (1944), Paré (1944), Celsus (1947), Rufus of Ephesus (1955), and Galen (1956). A few years ago at an annual meeting of the American Association of the History of Medicine, Ruiz Moreno was singled out for special honour. His wife, Luisa Allende de Ruiz Moreno, always accompanied him to international congresses, where the warmth of their personalities readily won them new friends.

JOHN WOTHERSPOON, M.D., Ch.B., D.P.H. D.T.M.&H.

Dr. J. Wotherspoon died at Beckenham Hospital on June 16 in his 86th year.

John Wotherspoon was born in Glasgow in 1875. He studied medicine at Glasgow University, and gained various class honours in his year, including first prize in the Practice of Medicine and Senior University Medal in Clinical Surgery. He graduated M.B., Ch.B. in 1897, and was subsequently house-physician and house-surgeon at the Glasgow Western Infirmary, house-physician at the Glasgow Maternity Hospital, and junior and senior house-surgeon at the Glasgow Eye Infirmary. He served in the South African War with the Scottish National Red Cross Hospital. On returning to this country he held a number of hospital appointments in London and proceeded M.D. Glasgow (commended) in 1905. He then settled in general practice at Brockley. He was a Territorial and served throughout the first world war in the R.A.M.C. After the war he returned to his practice at Brockley and took the D.P.H., London, in 1919. Wotherspoon retired from practice in 1940, but later that year resumed general practice in Beckenham, where he had been living for some years. He finally retired from medical practice in 1950, at the age of 75. In 1951 he took the D.T.M.&H. before setting out on a long-awaited holiday in South Africa and Rhodesia.

Dr. Wotherspoon was married and is survived by his younger son, also a doctor, and five daughters.

H. A. FREEMAN, M.R.C.S., L.R.C.P.

Dr. H. A. Freeman, who practised in Chester, died in the Royal Infirmary there on June 7 at the age of 52.

Harold Abraham Freeman was the son of the Rev. I. Freeman, and was born, brought up, and educated in Liverpool. He completed his medical training at the Liverpool School and qualified in 1937. After house appointments at Liverpool Royal Infirmary he went to the Chester City Hospital as a resident, where, apart from a short period in the Army, he remained until 1950, latterly as deputy medical superintendent. After the inception of the National Health Service there was no scope for this post, and so he started practice in Chester and near-by Hawarden, where he continued until his death, latterly in partnership.

Dr. Freeman was well known apart from his medical work. He had a very keen appreciation of music, and had been chairman of the Chester and North Wales Music Society since its formation. He was also a former chairman of the Chester Film Society and a vice-president of the Chester Theatre Club. Through all these activities he had a wide circle of friends, which was evident from the large congregation that gathered at his memorial service.

Harold Freeman was a man of great integrity and high ideals. It was almost impossible to get him to criticize anybody adversely, and gossip was anathema to him. Blessed with an attractive personality, he was loved by his patients, all of whom, rich and poor alike, he treated with great kindness, courtesy, and sincerity. He had a most distressing final illness, and although he struggled desperately to live the odds against him were too great, and after five weeks of severe suffering he passed away.

His death is a tragic loss for very many people, and the sympathy of everyone who knew him goes out to his widow. —A. C. S.

DOROTHY LANCASTER, M.B., B.S.

Dr. Dorothy Lancaster (née Godden), consultant dermatologist to the Caernarvonshire and Anglesey Hospital, died suddenly at her home in Bangor on May 7. She was 61 years of age.

Dorothy Lancaster received her medical education at the London School of Medicine for Women and St. Mary's Hospital, and after qualifying in 1926 spent a year in Vienna studying dermatology under Professor Oppenheim. She also worked with the dermatologist Dr. Kenneth Wills, of Bristol. In 1922 she married Mr. Leonard Lancaster, who is consultant surgeon to the Caernarvon and Anglesey Hospital, and went to live in Bangor. She was the only physician in North Wales at this time with special experience in dermatology. From 1946 onwards she was consultant dermatologist to the Caernarvonshire and Anglesey Hospital. Not only was she the first woman consultant to be appointed to the hospital but she founded a new department, the first in North Wales. This expanded to provide a comprehensive service in dermatology extending over a wide area. She was elected to a similar appointment in the Clwyd and Deeside area.

Dr. DORIS ODLUM writes: Dr. Dorothy Lancaster was a keen supporter of the activities of medical women and an active member of the Medical Women's Federation and secretary and president of the North Wales branch. She was also a member of the International Federation of Medical Women, and with them travelled to Canada and the U.S.A., where she also made an investigation into the skin manifestations of general disease in those countries. She delivered an address on skin diseases in women at the Medical Women's International Conference in Italy in 1954.

The welfare of nurses was one of her special interests, and she was president of the Caernarvon and Anglesey branch of the Royal College of Nursing and of the Caernarvon and Anglesey Hospital Nurses' Association. A lecturer and commandant of the Bangor detachment of the Red Cross, she was also medical officer of the Bangor Red Cross blood transfusion service. She was president of the Bangor Inner Wheel and also of the Bangor and district Soroptimist Club, and gave much of her spare time to furthering the interests of working women of many professions. She was also an accomplished needlewoman and especially enjoyed tapestry work. In her few hours of leisure nothing pleased her more than pottering about in her garden. She had a great capacity for friendship and kept open house.

Her sudden death has been a great loss to all her friends and devoted patients. I personally knew her well as a member of the Medical Women's Federation and the Medical Women's International Association, and travelled with her to many of their conferences. She was a delightful companion and a most loyal and devoted friend. When I was a patient in Bangor Hospital under the care of her

husband she showed me the greatest possible kindness. Our deep sympathy will go to Mr. Leonard Lancaster, and all of us who had the privilege of knowing her will grieve for her untimely loss.

JAMES WILSON-YOUNG, M.B., Ch.B., L.M.

Dr. James Wilson-Young, who practised for 35 years in Workington, Cumberland, died at his home near Carlisle on June 3. He was 66.

James Wilson-Young started his medical career at Glasgow in 1914, but on the outbreak of war he joined the Royal Scots Fusiliers, serving as a combatant officer in France and Egypt. In 1917 he was seconded to the R.F.C., in which he served as a pilot in Egypt and Palestine. At the end of the war he recommenced his medical studies and graduated M.B., Ch.B. at the University of Glasgow in 1924. In 1925 he set up in practice in Workington, Cumberland, where he remained for the rest of his professional life, retiring in May, 1960. In Workington he was on the staff of the local Infirmary, practising general surgery and gynaecology. He also was the doctor in charge of Workington Fever Hospital. He retained his interest in the armed Forces as Admiralty surgeon and agent, and in the second world war he commanded and trained a mobile civil defence unit. In 1950 he became a county magistrate and in 1958 he was appointed High Sheriff of Cumberland.

Wilson-Young led an active professional and social life. He was a countryman at heart, and loved riding and hunting and country shows. His home was the centre of social life in the area, and there his many friends were brought together to enjoy his rich hospitality.

As a doctor he was independent-minded and relied on his own clinical judgment. He knew all his patients intimately and was rarely wrong in his judgment. He instinctively brought comfort and support to a sickroom, for his very presence was therapeutic. A tall handsome man with a deep authoritative voice and manner, he quickly won the loyalty and affection of his patients.

The heart of his life was his home, and his many friends will always treasure the memory of the rich family life he enjoyed with his wife and children. His long and painful final illness he bore with courage and patience. He is survived by his widow, a son, and three daughters. His elder brother, Major-General Thomas Young, R.A.M.C. (ret.), was formerly Director of Army Health at the War Office.—J. S.

C. V. PATRICK, M.B., Ch.B., F.R.C.S.

Mr. C. V. Patrick, senior surgeon at the Royal Hospital, Wolverhampton, and surgeon in charge of the urological department there, died at his home in Wolverhampton on July 7.

Conrad Vincent Patrick won a university scholarship from Caius College, Cambridge, to St. Thomas's Hospital, where he won numerous medals and prizes. After qualification in 1922 he held a long series of resident appointments at St. Thomas's, including finally that of resident assistant surgeon. Having graduated M.B., Ch.B. at Cambridge in 1923 and taken the F.R.C.S. in 1924, he accepted the post of surgical registrar at the Royal Hospital, Wolverhampton, to the honorary staff of which he was soon elected. Thereafter he became one of the best-known surgeons in the area, holding numerous hospital appointments, serving on many professional committees, and lecturing in clinical surgery at the University of Birmingham. From 1941 to 1943 he was president of the Staffordshire Branch of the B.M.A.

Mr. Patrick is survived by his widow.

Mr. HENRY B. YOUNG writes: Others could write more formally of the death of Mr. Conrad Vincent Patrick, senior surgeon of the Royal Hospital, Wolverhampton, but may I, as one of his junior colleagues, add a few notes? By his passing, Wolverhampton has lost an outstanding surgeon, countless patients a friend, and the younger surgeons a wise counsellor and inspiring example. "C. V. P.," as he was known, was tireless in the pursuit of the welfare of his patients, and demanded the highest standards and most exacting discipline from his juniors. This dedicated "patient care" brought ceaseless demand on his time and energies from all classes over a wide area, but such was his energy and drive that he was able to meet all these needs with outstanding success. He was always keen, interested, and eager to try new procedures, a great stimulus to those around. A skilful determined operator, who over the years had dealt with all the specialties, he settled down to urology as his main surgical hobby. His high standard and progressive outlook and the width of his operative scope brought him young men in training in a steady stream. To those who measured up to his standard he proved a staunch friend, ever ready to take endless trouble to further their careers.

Despite his heavy surgical commitments in the area he managed to give time to the affairs of the region, and was chairman of the advisory committee in surgery; in this capacity he helped to ensure that in the partnership between region and teaching hospitals the region took its place. In committee he was an excellent, knowledgeable, and business-like member or chairman.

At home, Mr. and Mrs. Patrick were the ideal hosts for their many friends, and the happy and animated gatherings on the lawn of his beautiful garden will always be remembered. Outside his work he was widely read, and keenly interested in golf, bee-keeping, and gardening. In his younger days he had raced at Brooklands and always retained his interest in fast though elegant cars.

It is unbelievable that we will never see the purposeful characteristic figure of C. V. P. in the corridors, meetings, or in reassuring support at the bedside again. Even the manner of his end was characteristic of his life, in that, knowing his days were numbered, he tidied up his affairs with his usual personal care and attention. He lived fully and well and could have no regrets, and those who have been fortunate enough to know him have been enriched by their fellowship with him.

PHILIP R. PETERS, M.R.C.S., L.R.C.P.

Dr. Philip Raymond Peters died at the age of 43 years on June 18 at St. Bartholomew's Hospital. He had worked with Moral Re-armament in Asia, Europe, and America.

Dr. L. C. MONTGOMERY writes: Philip Peters studied at Caius College, Cambridge, and Sheffield, qualifying M.R.C.S., L.R.C.P. in 1943. After a year in Rotherham Hospital he went to Burma in the R.A.M.C. For Philip life meant people, and medicine was never limited to the physical and the pharmaceutical but was also intended to heal the hurts and hates in human lives and liberate men for whole-hearted, selfless, vibrant living. He would have settled in Sheffield after the war. He had many friends there; but he said, "It would be tragic if any of us were to decide the future on a basis of personal security." He gave his interest to the national post-war problems of shortage of coal and steel and to the bitterness which contributed to it. Very soon he was again in Asia, travelling in response to invitations from leaders of India, Pakistan, Ceylon, and Burma who had asked for Moral Re-armament to come to their countries. Philip was often the pioneer in these situations, meeting local people, finding hospitality, theatres, transport. It was an arduous life subject to hardship, privations, and infections. He did not think of this aspect personally but was increasingly aware of the march of materialist tyranny. To provide the inspired moral answer in men's lives was his passion and commitment. He gave his life for the moral re-armament of nations. He was one of those who helped to create in Kerala the unity which made it possible for the democratic forces—Hindu, Christian, and Muslim—to defeat the Communists

at the last election. Only four weeks before he died from leukaemia he was standing on the pavement in London to give a hearty welcome to five leading Burmese Buddhist abbots; and when later their leader heard at Caux of Philip's passing, he said, "I spent the morning praying for him. He was a great man."

JULIAN TAYLOR, C.B.E., M.S., F.R.C.S. Hon.F.R.A.C.S.

The obituary of Professor Julian Taylor was published in the *Journal* of April 29 (p. 1255).

Dr. I. A. NABRI writes: The obituary of Professor Julian Taylor in the Sudan newspaper *The Sudan Daily* speaks for itself, but I should like to add that it is beyond doubt Professor Taylor has left in the Sudan friends who are as sincere, though not as many, as his friends in Britain and other countries. His loss has been deeply felt by the many "junior" doctors, of whom I am one, for whose betterment he spared neither time nor energy. We have not only lost a great friend but also a great teacher and father.

H. J. SHORVON, M.B., B.S., D.A., D.P.M.

The obituary of Dr. H. J. Shorvon was printed in the *Journal* of May 27 (p. 1546).

Dr. K. C. DUBE, superintendent of the Mental Hospital, Agra, India, writes: I learnt with deep sorrow of the death of Dr. H. J. Shorvon from the obituary columns of your journal. I had the privilege of working with Dr. Shorvon in Belmont Hospital, Sutton, in 1949, and I can very vividly remember him as a man of outstanding ability with a great drive for work. He was deeply interested in the welfare of his patients and worked incessantly to understand his cases in the quickest possible time. I have yet to see a psychiatrist who would come to grips with his patient's problem in such a short time. He was very successful with abreactive treatment, and it was a pleasure to watch him at work. A charming man—helpful, cheerful, and conscientious—he will always be remembered by those who met him. His death is a great loss to psychiatry.

H. G. ABERCROMBIE, M.D., D.T.M.

The obituary of Dr. H. G. Abercrombie was published in the *Journal* of June 10 (p. 1690).

F. B. writes: I was a member of a party including the late Dr. H. G. Abercrombie which travelled to Moscow in 1934 to an international congress on rheumatism. Dr. Abercrombie was much the most senior member of the party. As the train travelled slowly through Poland, stopping at wayside stations to take in water, Dr. Abercrombie used to descend from his coach and wander along the cuttings botanizing. The engine-driver would give a warning toot and we would see Dr. Abercrombie some way down the line. Sir Arthur Porritt and myself would sprint down the track, seize the botanist by each elbow, and race him back to the now-accelerating express and heave him on to the platform of the observation car at the end of the train. This happened more than once, and each time Abercrombie thanked us breathlessly, and then went on to instruct us in the beauties of the specimens he had collected.

GEORGE DALZIEL, *M.C.*, M.B., Ch.B., F.R.F.P.S.

The obituary of Mr. George Dalziel was published in the *Journal* of July 8 (p. 117).

Mr. KENNETH FRASER writes: Mr. George Dalziel's surgery was characterized by an unusually high degree of manual dexterity. He was particularly skilled in abdominal surgery and in the treatment of fractures. At all times most considerate and helpful to his assistants and students, George Dalziel had that great quality of encouraging his assistants to carry out original work, and in equal measure he derived great pleasure from the resultant surgery carried out by them in his wards. His patients loved him, for he was well known for his kindly disposition, and he will be greatly missed.

MARCEL JUNOD, M.D., F.F.A. R.C.S.

The obituary of Dr. Marcel Junod was published in the *Journal* of July 8 (p. 116).

T. H. S. writes: Marcel Junod, who died last month in his own hospital in Geneva, was a man whose visits to this country were welcomed to the full by all those who knew him. His period of study in anaesthetics, undertaken after the rigours of his war work for the Red Cross, brought him into contact with many people here who are proud to regard themselves as his friends. Junod's charity and capacity for friendship were characteristics which made him one of the most valuable workers in the International Red Cross. During the war he visited every combatant country; he argued, negotiated, and struggled to help the lot of many thousands of those unfortunate enough to be victims of war, and he earned the respect and confidence of all. He would recount how his duties gave him the unique and doubtful privilege of being bombed in every combatant capital in Europe, with some in Asia thrown in for good measure.

He was a truly delightful and great character: modest, unassuming, and humorous in his personal relationships, but relentless and fearless in pursuit of any matter of principle that would help mankind.

Medico-Legal

CONSULTANTS' TAXES: TEST CASES
HOUSE OF LORDS DECISION

[FROM OUR LEGAL CORRESPONDENT]

The House of Lords has decided in favour of the Crown in five test cases selected by agreement between the B.M.A. and the Inland Revenue in order to decide the tax position of medical practitioners who hold appointments as part-time consultants under the National Health Service and also carry on private professional practice (*The Times*, July 7). In so deciding, the House of Lords reversed the decision of the Court of Appeal (*Journal*, April 2, 1960, p. 1062).

The five consultants were Dr. H. L. Ross, consultant radiologist; Dr. A. Hirtenstein, consultant ophthalmologist; Dr. A. G. Marshall, consultant pathologist; Dr. H. P. Tarnesby, consultant psychiatrist; and Mr. C. E. Drew, consultant thoracic surgeon. During the relevant years of tax assessment all these consultants held part-time appointments under the National Health Service and also had private patients.

The question for the courts in these cases was to decide the classification of the income of these part-time consultants under Schedule D or Schedule E. Schedule D is the more attractive to the taxpayer because of its more liberal permissible deductions for expenses. By the time the cases reached the House of Lords it was conceded that the income received from private practice fell under Schedule D and the income from part-time National Health Service appointments fell under Schedule E with its narrower definition of permissible allowances. The Court of Appeal, however, had decided that in computing his liability under Schedule D a consultant was entitled to include in his deductions the expenses incurred in his National Health Service appointment to the extent that they were not allowed under Schedule E.

Counsel for the consultants were concerned before the House of Lords to support this decision of the Court of Appeal. The House of Lords rejected their arguments. Their Lordships held that it was fundamental law that the schedules to the Income Tax Acts were mutually exclusive: each schedule with its related rules formed a complete code for dealing with the income falling under it, and dealt with

allowances, deductions, or exemptions relating to them respectively. In fact, it was not possible to deduct under one schedule (in whole or in part) expenses incurred in respect of earnings falling under another schedule. The income of these consultants was divided between Schedule D and Schedule E: to determine the consultant's tax liability it was not permissible to look further than the rules under each schedule to determine his liability under each.

The B.M.A. in conjunction with certain other bodies, including the three medical defence organizations, have supported the five consultants throughout in their appeals and will pay the costs.

Medical Notes in Parliament

HOSPITAL ADMISSIONS UNDER MENTAL HEALTH ACT

[FROM OUR PARLIAMENTARY CORRESPONDENT]

Lady WOOTTON, seeking information about the operation of the Mental Health Act, asked the Government on July 4 for figures of the admission to hospitals of psychopaths and of the mentally disordered. It appeared from the answers that so far only the figures for November and December, 1960, are available, and that it will be some time before figures for the first six-month period will be so.

Psychopaths and the Mentally Disordered

The totals for the two months in England and Wales which were given her were: persons suffering from psychopathic disorder admitted to hospital (Part IV), 38; persons similarly classified subject to hospital orders made by the courts (Part V), 16; none were received into guardianship during this period. Over the same two months 19,523 mentally disordered persons were admitted to hospital on an informal basis, and there were 4,901 compulsory admissions. Lady WOOTTON deferred further questions until later figures were available. Lord TAYLOR commented that the number of compulsory admissions of the mentally disordered seemed rather high, and was told by Lord NEWTON, the Government spokesman, that 4,127 of them were for observation (for periods not exceeding 28 days) and only 774 for longer periods of treatment.

Regional Figures

These totals were broken down into hospital regions, as shown in the following Table.

Region	Psychopaths		Mentally Disordered	
	Part IV	Part V	Informal	Compulsory
Newcastle	3	—	1,266	259
Leeds	2	—	1,302	279
Sheffield	5	2	1,431	406
East Anglia	—	1	579	136
Metropolitan:				
N.W.	8	—	1,440	414
N.E.	—	—	1,111	218
S.E.	1	2	1,606	353
S.W.	2	1	2,695	538
Oxford	2	1	520	129
South-West	5	4	1,271	308
Wales	1	—	1,158	235
Birmingham	3	2	1,891	505
Manchester	1	1	1,397	314
Liverpool	4	—	1,020	533
Wessex	1	—	836	255
Special hospitals		2	—	19
Total	38	16	19,523	4,901

QUESTIONS IN THE COMMONS

Hospital Pharmacists

The MINISTER OF HEALTH informed Sir HUGH LINSTEAD (Putney, Con.) on July 3 that the numbers of pharmacists (expressed in terms of whole-time equivalents) in the hospital service at the end of each year from 1949 to 1960 were: *1949*, 1,127; *1950*, 1,176; *1951*, 1,217; *1952*, 1,243; *1953*, 1,242; *1954*, 1,237; *1955*, 1,267; *1956*, 1,305; *1957*, 1,314; *1958*, 1,315; *1959*, 1,348; *1960*, 1,444.

Accidents in the Home

Dr. DONALD JOHNSON (Carlisle, Con.) asked the Minister of Health on July 4 for the number of deaths of people injured by accidents in the home in 1960, and the principal causes of death.

Miss EDITH PITT, Parliamentary Secretary, gave him the following figures of deaths from accidents in England and Wales.

	Males	Females
Coal-gas poisoning	391	526
Other poisoning	160	212
Falls	1,199	3,027
Burns and scalds	221	432
Choking and suffocation	285	226
Other accidents	220	129
All accidents	2,476	4,552

Assistance Granted on Prescription Charges

Mr. KENNETH ROBINSON (St. Pancras North, Lab.) asked the Minister of Pensions and National Insurance on July 7 to state the number of prescription charges refunded by the National Assistance Board, and the sums involved, for each month of this year for which figures were available, distinguishing if possible between refunds to those in regular receipt of national assistance and others.

Miss PAT HORNSBY-SMITH, Parliamentary Secretary, stated that the total amounts of national assistance in respect of charges for prescriptions under the Health Service which had been paid this year up to the month of April were: January, £100,000; February, £96,000; March, £213,000; April, £177,000. It was not possible to say for all these figures how many prescribed items the amount represented. A single refund was commonly claimed for charges paid over a period, and consequently the March figure, and probably the April figure to a less extent, included some refunds of charges at the former rate of 1s. per item as well as charges at the current rate of 2s. Detailed information about this was not available. Firm figures as regards refunds to persons not receiving a regular weekly allowance were available only for those of them who were not provided for under special arrangements for residents in old people's homes and the like. These were:

	No. of Grants	Total Amount Granted
7 weeks to January 31	3,915	£1,139
4 ,, ,, February 28	2,930	£833
4 ,, ,, March 28	6,138	£2,114
4 ,, ,, April 25	11,221	£3,838
5 ,, ,, May 30	19,588	£6,921

Mr. ROBINSON also asked how many applications for refund of Health Service charges had been rejected by the National Assistance Board. Miss HORNSBY-SMITH stated that the numbers of unsuccessful applications recorded this year up to May were: in seven weeks to January 31, 2,914; in four weeks to February 28, 2,662; in four weeks to March 28, 3,876; in four weeks to April 25, 3,953; and in five weeks to May 30, 4,578.

The Inter-Church Committee on the Ministry of Healing has published three pamphlets in its Christian Family Series: No. 1, *For Those Who Are Having a Baby*; No. 2, *For Those in Need of Comfort*; and No. 3, *When Labour Begins and Baby Arrives*. The pamphlets cost 3d. each, and inquiries should be sent to the Committee at 9 Osborne Avenue, Newcastle upon Tyne 2.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

The following candidates have been approved at the examinations indicated:

FINAL M.B.—*Part II:* [1 2 3]P. W. Adams, [1 2]E. J. Anderson, [1 2 3]D. R. Aubrey, [1 3]P. R. H. Barbor, [2 3]R. S. Bartholomew, [1 2 3]N. M. Bassett, [1 3]J. S. M. Beales, [1 2 3]P. G. Beauchamp, [2]R. J. E. Bellars, [1 2 3]M. Bewick, [1]C. C. Black, [1 3]R. D. Blacklidge, [1 2 3]D. I. Bowen, [1 2 3]J. E. Bowen, [2 3]N. R. H. Boyd, [1 2 3]J. B. Brown, [1 2 3]S. E. Bundey, [1 2 3]G. T. Bungay, [1 2 3]J. K. Butler, [1 2 3]D. W. Cairns, [1 3]P. B. Carslake, [1 2 3]S. Channell, [1 2 3]R. H. G. Charles, [1 2 3]J. R. Chesterton, [1 2 3]J. B. Cocking, [1 2 3]N. K. Coni, [1 2 3]C. K. Connolly, [1 2]D. Cook, [1 2 3]F. B. Cookson, [1 2]P. E. Copus, [1 2 3]A. G. L. Corkill, [1 2 3]A. V. Cowan, [1]B. R. M. Crook, [1]J. O. Crosse, [1 2 3]A. G. O. Crowther, [1 2 3]R. J. Cryer, [1 2 3]J. S. Cunningham, [2 3]J. W. Dale, [1 2 3]C. Dare, [1 2 3]M. R. E. Dean, [1]F. T. de Dombal, [1 2]R. S. Deraniyagala, [1 2]J. W. D. Drake-Lee, [1 2 3]R. A. Durance, [1 2 3]N. H. Dyer, [1 2 3]G. M. Eames, [1 2 3]A. M. Edwards, [3]R. H. Eleady-Cole, [1 2 3]D. E. Elgar, [1 2 3]D. J. Evans, [1 2 3]J. G. Evans, [1 2 3]A. N. Fawcett, [1 2 3]W. R. F. Ferguson, [2 3]E. S. Field, [1 2 3]H. L. Firth, [1 2 3]H. M. Fischer-Webb, [1 2 3]J. A. Fixsen, [1 2 3]O. J. Follows, [1 2 3]G. J. J. Fuzzey, [2]G. W. Gamlen, [1 2 3]J. R. C. Gardham, [1 2 3]D. Gardner-Medwin, [1 2 3]D. W. Gau, [1 2 3]D. G. Gibson, [3]A. V. Gillespie, [1 2 3]C. M. T. Gleave, [1]J. F. A. McC. Graham, [3]W. D. Graham-Brown, [1 2]K. E. Gray, [1 2 3]F. R. Greenlees, [2]G. Hamber, [3]J. W. Hamilton, [3]I. W. F. Hanham, [1]P. S. E. G. Harland, [2 3]P. W. Harvey, [1 2 3]C. P. Heanley, [1 2]J. M. Henderson, [1 2 3]R. L. Himsworth, [2 3]N. E. H. Holmes, [1 2 3]E. M. Holt, [1 2 3]R. J. Hope, [1 2 3]W. N. Hutton, [1 2 3]D. M. Hynes, [1 2 3]N. Jankey, [1]H. Jay, [1 3]D. M. Jennens, [1 2 3]A. E. Jephcott, [1]R. A. Johnson, [1 2 3]E. W. L. Jones, [1 2 3]T. G. Jones, [1 2 3]F. R. Jordan, [1 2 3]W. J. Jory, [1 2 3]A. P. Joseph, [1 2 3]J. E. A. Knowles, [1 2 3]H. H. M. Knox-Macaulay, [1 2]R. C. Lallemand, [1 2 3]D. D. Law, [1 2 3]S. Y. Lee, [1 2 3]J. Lendrum, [1 2 3]G. D. Loft, [1 2 3]I. L. Mackenzie [2]R. K. Mackenzie Ross, [3]G. I. M. McLellan, [1 2 3]N. C. Macmillan, [1 2 3]J. R. Maltby, [1 2 3]A. P. Manasse, [1 2 3]R. W. Marcuson, [1 2 3]J. R. Mariar, [1]C. H. Maycock, [1 2]J. J. Meanley, [1 2 3]A. J. B. Missen, [1 2]J. H. Moran, [1 2 3]M. C. W. Morgan, [1 2 3]J. O. Morgan-Hughes, [1 2]M. T. Morrell, [1]P. H. W. Mylechreest, [1 2 3]C. W. Newrick, [1 2 3]J. B. Newth, [1 2 3]P. J. Noble, [1 2 3]W. J. Norman, [1 2 3]S. Nundy, [1 2 3]I. Oates, [1 2 3]S. E. Odamtten, [1 2 3]W. P. Ormerod, [1 2 3]D. H. Orrell, [1 3]B. P. N. Osler, [1 2 3]J. P. A. Page, [1 2 3]J. W. Partridge, [1 2 3]W. M. P. Patterson, [1 2]H. J. Pendleton, [1]S. P. B. Percival, [1 2 3]J. Phillips, [1 2 3]G. Pincherle, [1 2 3]D. M. Potts, [1 2 3]T. D. Preston, [1 2 3]R. D. Pyrah, [1 2 3]P. H. Rees, [1 2 3]A. B. Richards, [1]C. H. L. Rigby, [1 2 3]M. T. S. Roberts, [1 2 3]A. P. Rubin, [1 2 3]J. E. L. Sales, [1 2 3]M. N. Sampson, [1 2 3]J. H. Sandford-Smith, [1 2 3]K. B. Saunders, [1 2 3]G. L. Scott, [1 2 3]P. V. Scott, [1 2]S. L. Sellars, [1 2 3]W. S. Shand, [1 2 3]E. H. Shaw, [1 2 3]J. Shenkman, [1 2]M. A. Shirley, [2]The Hon. T. C. B. Stamp, [2]B. J. Stoodley, [1 2 3]M. C. Storey, [1 2]M. F. Sullivan, [1 2 3]J. E. O. Sunkwa-Mills, [1 2]M. Tarrant, [1 2 3]A. B. W. Thomas, [1 2]C. Tremlett, [1 2 3]G. E. R. Vaughan, [1 2 3]E. R. Verrier-Jones, [1]P. R. J. Vickers, [1]C. D. Walker, [1 2 3]P. J. Watkins, [1 2 3]R. H. Weeks, [1 2]P. B. Wemyss-Gorman, [1 2 3]J. A. B. Weston, [1 2]O. C. Wetherell, [1 2 3]D. G. Wilkins, [1 2 3]H. T. Williams, [1 2 3]J. L. Williams, [1 2 3]E. B. Wilson, [1 2 3]A. D. Wright, [1 2 3]K. S. Young, [1 2 3]I. Ziment. *Part I (Pathology and Pharmacology):* N. R. H. Boyd, S. E. Bundey, B. R. M. Crook, J. O. Crosse, A. G. O. Crowther, M. R. E. Dean, A. J. Gordon, P. W. Harvey, J. M. Henderson, A. D. Holt-Wilson, R. J. Hope, B. J. Hyde, R. A. Johnson, F. R. Jordan, A. P. Joseph, R. J. Lavin, C. H. Maycock, J. H. Moran, C. W. Newrick, I. Oates, W. P. Ormerod, B. P. N. Osler, Mrs. M. J. B. Ransom, C. H. L. Rigby, W. S. Shand, B. J. Stoodley, M. F. Sullivan, P. B. Wemyss-Gorman.

[1]Passed in principles and practice of physic. [2]Passed in principles and practice of surgery. [3]Passed in midwifery and gynaecology.

UNIVERSITY OF GLASGOW

Dr. J. F. Lamb has been appointed a Lecturer in Physiology.

Dr. I. A. Carr, Lecturer in Pathology at Glasgow Royal Infirmary, has resigned on his appointment as Senior Lecturer in Pathology in the University of New South Wales.

On June 24 the degree of M.D. was conferred on A. B. McL. Currie, G. L. Gibson, and Maria A. Ratzer.

The Diploma in Public Health was awarded to the following candidates on June 20:

F. Abidzadeh, Marjory M. Buchanan, W. Ducat, N. Fakhar, Emily A. Friel, F. C. Garry, P. J. Halket, H. MacAnespie, Sarah C. McEwan, Jean M. McEwen, T. L. O'Donnell, S. G. Phillips.

UNIVERSITY OF DUBLIN

SCHOOL OF PHYSIC, TRINITY COLLEGE

The following candidates have been approved at the examinations indicated:

DIPLOMA IN PSYCHOLOGICAL MEDICINE.—*Part II:* A. A. Marinho.

DIPLOMA IN GYNAECOLOGY AND OBSTETRICS.—S. Akhter, D. H. Bailey, Y. M. Jadhav, A. A. Shaikh, S. Sinnarkar.

On June 29 the following degrees were conferred:

M.D.—R. C. Gray, Barbara W. M. Partridge.

M.B., B.CH., B.A.O.—Doreen J. Aucott, W. H. Beesley, T. J. S. Black, C. E. Boursin, R. M. Bridburg, J. E. Cahill, C. J. Condron, J. M. S. Cussen, J. H. Detheridge, K. H. Du Plessis, D. E. Dyer, G. B. R. Fisher, D. J. FitzPatrick R. M. Galloway, J. E. Gibbon, Anne M. Gillett, W. J. W. Greene, Jane A. W. G. Johnston, V. H. Keely, J. H. M. Lyndsay, M. E. McGrath, Bernardine O'Neill, G. Patrikios, M. B. R. Roberts, Jennifer A. Shannon, R. Sinniah, N. J. Shelley Smith, T. Stack, Maria J. Strzelecka, A. O. Williams.

L.M., L.CH., L.A.O.—D. J. McKay.

UNIVERSITY OF LONDON

Dr. W. K. Taylor has been nominated for appointment, and Dr. G. W. Heckels renominated for appointment, as representatives of the University on the governing bodies of the George Abbot County Secondary School, Guildford, and the Rosebery County School for Girls and Glyn County School for Boys, Epsom, respectively.

The following have been recognized as teachers of the University in the subjects indicated in parentheses: *St. George's Hospital Medical School*, Dr. J. C. Batten (Medicine), Dr. D. D. C. Howat (Anaesthetics), Dr. Harold Stern (Pathology (Virology)), Mr. L. S. Walsh (Surgery), and Mr. G. C. Lloyd-Roberts (Orthopaedics). *Guy's Hospital Medical School*, Dr. D. C. Deuchar (Medicine (Cardiology)) and Dr. J. M. Hall (Anaesthetics). *Westminster Medical School*, Mr. R. D. de Vere (Obstetrics and Gynaecology). *Royal Free Hospital School of Medicine*, Mr. J. P. Hopewell (Surgery). *St. Thomas's Hospital Medical School*, Mr. H. E. Lockhart-Mummery (Surgery) and Dr. J. W. Pierce (Radiology). *Postgraduate Medical School of London*, Dr. Iain MacIntyre (Chemical Pathology) and Dr. J. P. Shillingford (Medicine). *Middlesex Hospital Medical School*, Dr. B. A. Sellick (Anaesthetics).

The A. H. Bygott Scholarship for 1960–1 has been awarded to Dr. Athol Noble Hepburn, and the Sambrooke Scholarship in Medical Science, at King's College, to J. G. Williams (Wolverhampton Grammar School).

On May 17 the degree of M.D. was awarded to S. J. Beales, N. S. Capstick, and R. H. C. Wells.

On May 8 the degree of Ph.D. in the Faculty of Medicine was awarded to D. M. Shaw, M.B., B.S. (London Hospital Medical College and King's College), and on May 31 the same degree was awarded to A. J. Evans (School of Pharmacy), H. A. Ghaleb (School of Pharmacy and Institute of Cancer Research: Royal Cancer Hospital), M. B. Ibrahim (Guy's Hospital Medical School), and Margaret E. H. Turner Warwick, D.M., M.R.C.P. (Institute of Diseases of the Chest).

UNIVERSITY OF WALES

WELSH NATIONAL SCHOOL OF MEDICINE

The following candidates have been approved at the examination indicated:

M.B., B.CH.—Hilda Bailey, V. A. Church, R. H. Clark, R. A. Cope, [1 2]Deirdre J. Curran, D. M. Davies H. S. Davies, J. S. Davies, [2]Joan Evans, R. W. Fortt, Nancy E. Francis, [2]Alfred Kwong Chun Hui, P. D. L. John, [4]D. J. Jones, Helen W. Jones, [1 2]Margaret E. Jones, Anne M. Kriescher, D. G. Lewis, C. C. McCormick, [5]Wing Shang Ng, [3 4 5]D. K. Peters, Jean M. Peters, C. A. Quick, Jennifer H. Roberts, [4]K. J. Russell, Patricia M. Wardlaw, J. G. Williams, N. B. Williams, [6]A. C. MacD. Windsor, B. Wookey.

[1]Distinction in Forensic Medicine and Toxicology. [2]Distinction in Social Medicine and Public Health. [3]Distinction in Surgery. [4]Distinction in Midwifery and Gynaecology. [5]Distinction in Pharmacology and Therapeutics.

UNIVERSITY OF LIVERPOOL

The following candidates have been approved at the examinations indicated:

M.D.—R. Finn, R. W. Galloway, R. Harris.

CH.M.—P. F. Lynch.

M.B., CH.B.—[1 4]Jean D. Ames, [2 3]D. W. Fielding, B. R. Anderson, A. K. Boaitey, I. G. Bogle, D. Canter, Ann F. Capey, M. R. J. Claxton, S. B. Cohen, M. Cook, A. Cooper, Sheila M. Cooper, Dolores Corcoran, Josephine C. Corry, F. Cunliffe, E. C. Daniels, Betty Dickinson, Jane Edwards, Georgina A. Ferguson, J. M. Findlay, Marie C. Firth, B. Garston, Pamela M. Hagan, B. V. Harris, Judith A. Heyworth, B. T. Higgins, Joan C. Higgins, J. D. L. Holme, E. W. Humphreys, J. E. Hutson, D. M. Johnston, R. O. Jones, A. P. Kenna, J. M. Kingston, Sheila Lang, T. Langan, [4]Dorothy A. Langton, I. K. Lewkonia, [4]Mary B. Lowe, G. A. McGregor, Janine M. Miller, E. C. Moore, Marjorie Moorhouse, T. M. Murphy, E. G. Parkinson, Aideen D. C. Perry, P. R. Prideaux, A. V. Pullin, Moira Ravey, Anne W. Rees, Patricia M. Ronald, L. Rosenbloom, G. Ryan, Freda Sanderson, [4]A. H. Short, A. Slater, Ethel V. Spence, P. H. Stevens, Annette Tattersall, R. Underwood, Gabriel M. Walsh, K. White, H. G. M. Williams, E. Wilsher, Jean J. Wilson, R. N. Wynn-Williams. *Passed in separate subjects:* J. Burns and D. Carrigan (medicine and surgery); H. I. Davies, T. France, and L. R. Priaulx (medicine and obstetrics and gynaecology).

DIPLOMA IN PUBLIC HEALTH.—A. Abdulmawjoud, D. A. Akintonde, J. H. E. Baines, M. A. Bari, N. A. De Heer, A. Griffith, Mahameidevi Gunawardena, W. N. O'Shea, L. J. Perez-Rios, Elizabeth J. Sutton.

DIPLOMA IN RADIOLOGY.—*Radiodiagnosis:* H. D. C. Bell, G. H. Evans, S. G. Groves, J. P. Lawson, A. McDermott, I. F. Quayle, Mary C. Wood. *Radiotherapy:* S. M. Masood.

[1]With first-class honours. [2]With second-class honours. [3]Distinction in surgery. [4]Distinction in obstetrics and gynaecology.

UNIVERSITY OF LEEDS

The following candidates have been approved at the examinations indicated:

M.D.—M. S. Losowsky.

M.B., CH.B.—D. J. Ashelford, B. Ballantyne, P. J. Bretherick, J. Ellis, M. Klinger, J. K. Merritt, J. M. Raper, R. M. Riley, P. J. Smith, B. E. Walker, Pamela J. Walker, Syd Moy Wong.

DIPLOMA IN PUBLIC HEALTH.—J. E. Lee.

UNIVERSITY OF BRISTOL

The following candidates have been approved at the examinations indicated:

FINAL M.B., CH.B.—*G. J. A. Browne (with Distinction in Surgery), *A. A. Brzechwa-Ajdukiewicz, *Edith M. Creighton, *B. H. Cummins *A. G. Morgan, *J. A. Morphew, *Violet A. Payne, *Diana M. Young, D. W. R. Carr, A. M. Chrysaphinis, Marjorie A. Coutts, Aerona M. A. Crick, M. J. Crick, K. A. Day, A. B. O. Desalu, J. D. Egdell, I. F. Elliott, K. H. Gay, G. H. Gundy, C. P. Hallett, B. Hanstead, D. W. Hide, Eleanor J. A. Hughes, R. C. W. Hughes, N. Ikoku, W. A. Knight, J. M. McGarry, Vida V. Mungwira, A. S. Parker, R. F. Payne, M. F. Reed, R. R. Roy (with Distinction in Public Health), A. K. Smeeton, T. D. Waterhouse, M. R. Watts. *In Group I only:* D. A. O. Sutton. *In Group II only:* J. E. Barber, P. J. Froud, R. A. Jones, D. F. Kitching.

*With second-class honours.

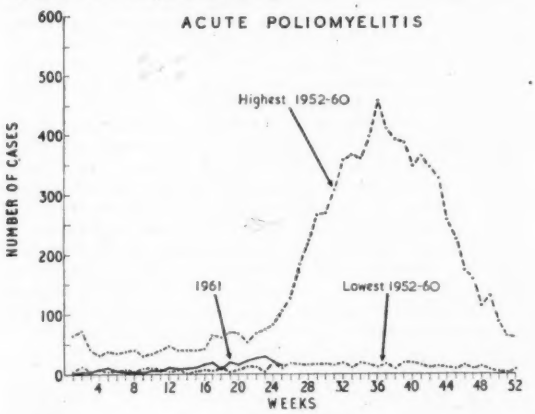
Vital Statistics

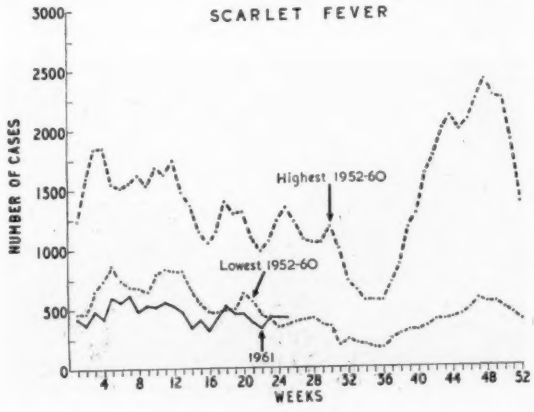
Poliomyelitis in the Rhineland

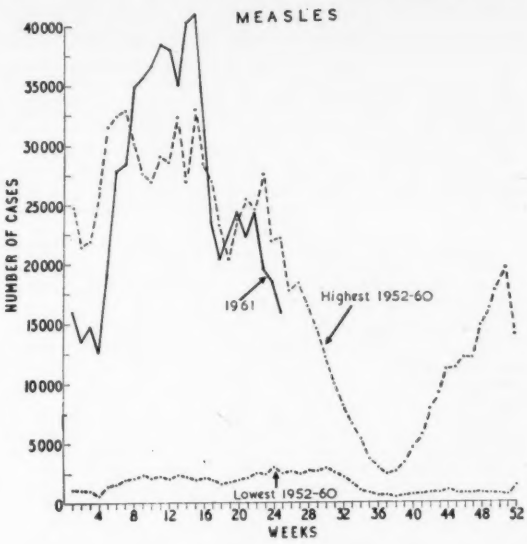
According to press reports, over 400 cases of poliomyelitis have been notified in the Rhineland, and there have been 21 deaths. The Federal Health Council has decided for the time being not to order a supply of the Sabin live vaccine, but to reconsider the matter in October after studying the results of tests carried out in Britain and in the U.S.A.—*Sunday Times*, July 9, 1961.

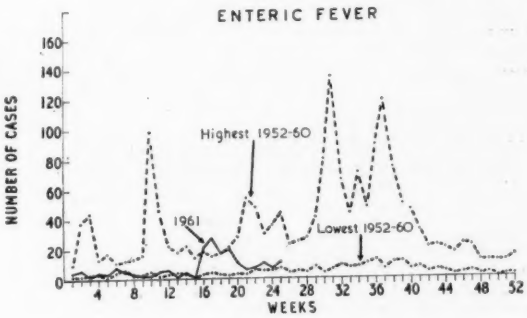
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported in each week during the years 1952–60 are shown thus ------, the figures for 1961 thus ——. Except for the curves showing notifications in 1961, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

Areas where the numbers of notifications were high in the latest two weeks for which figures are available.

	Week ending June 24	Week ending July 1
Dysentery		
Kent	13	**27**
Chislehurst and Sidcup U.D.	0	**16**
Staffordshire	23	**42**
Newcastle-under-Lyme M.B.	3	**12**
Newcastle-under-Lyme R.D.	0	**14**
Measles		
Devonshire	535	**495**
Plymouth C.B.	220	**202**
Gloucestershire	690	**913**
Bristol C.B.	208	**317**
Staffordshire	962	**910**
Stoke-on-Trent C.B.	348	**307**
Acute poliomyelitis		
Lancashire	3	**4**
Liverpool C.B.	1	**2**
Glamorganshire	1	**4**
Swansea C.B.	0	**2**
Food poisoning		
Lancashire	90	**44**
Kirkby U.D.	4	**13**
Surrey	5	**25**
Dorking and Horley R.D.	0	**20**

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **June 24** (No. 25) and corresponding week 1960.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the whole of England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES	1961					1960				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria ..	1	1	2	0		3	1	0	0	1
Dysentery ..	455	52	170	17	3	800	106	215	9	1
Encephalitis, acute	2	0		0		2	0	0	0	
Enteric fever:										
Typhoid ..	3	1	0	0		2	0	0	0	
Paratyphoid ..	8	0	2(B)	0		5	1	0	0	
Food-poisoning ..	257	35	24	4		265	25	21	5	
Infective enteritis or diarrhoea under 2 years ..				14	30				18	21
Measles*	15,864	420	278	290	127	2,465	134	28	4	1
Meningococcal infection	10	1	8	0		18	1	15	1	
Ophthalmia neonatorum	16	2	3	0		15	1	4	0	
Pneumonia† ..	154	12	129	0	4	173	10	140	5	1
Poliomyelitis, acute:										
Paralytic ..	10	0	0	5		7	0	0	0	7
Non-paralytic ..	4	0	0			2	0	0		
Puerperal fever‡ ..	146	17	14	0	1	152	31	13	0	
Scarlet fever ..	410	30	32	30	11	365	16	40	14	27
Tuberculosis:										
Respiratory ..	391	62	61	19		426	49	73	17	
Non-respiratory	41	5	12	5		43	4	7	6	
Whooping-cough	423	9	56	4	27	1,561	123	148	2	29

DEATHS	1961					1960				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria ..	0		0	0	0	0	0	0		0
Dysentery ..	2	0	0	0		2	0			
Encephalitis, acute		0			1		0			0
Enteric fever ..	0	0	0	0		0	0	0		
Infective enteritis or diarrhoea under 2 years	5	0	0	3	5	2	1	1		0
Influenza	6	0	1	0	0	5	0	0		0
Measles		0	0	0	0		0	0		0
Meningococcal infection		1	0				0	0		
Pneumonia ..	409	22	23	2	3	412	21	15		5
Poliomyelitis, acute	1	0		0	0	1	0			0
Scarlet fever ..	0	0	0	0	0		0	0		0
Tuberculosis:										
Respiratory ..	51	0	5	0	1	57	4	3		3
Non-respiratory		0		0	0		0	1		0
Whooping-cough	0		0	0	0	2	0	0		0
Deaths 0–1 year ..	295	31	26	10	9	293	31	37		9
Deaths (excluding stillbirths) ..	8,820	704	539	126	87	8,643	710	514		163
LIVE BIRTHS ..	16,084	1363	1005	273	396	15,396	1325	1088		438
STILLBIRTHS ..	283	24	19			292	24	19		

* Measles not notifiable in Scotland, whence returns are approximate
† Includes primary and influenzal pneumonia.
‡ Includes puerperal pyrexia.

Medical News

Colonial Territories Annual Report

The *Report on the Colonial Territories for 1960–61* (H.M.S.O., price 7s.) says that pressure on the curative medical services in the territories continued to be great. There was a substantial increase in the turnover of hospital in-patients and in the number of out-patients treated. While malaria, sleeping sickness, and yaws have, in many territories, been virtually eliminated or brought under control, this has served to highlight the residual problems. Tuberculosis constitutes a growing public health threat and undernutrition remains widespread. In many territories morbidity and mortality rates for intestinal and other infections resulting from defective sanitation continued to be high. The Oppenheimer College of Social Service, which is to train students of all races from each of the three territories of the Federation of Rhodesia and Nyasaland, has opened in temporary accommodation, and it is hoped to complete the first stage of the permanent building in Lusaka in the autumn. At the end of 1960 the number of colonial students in the United Kingdom and the Irish Republic was 15,292, a net increase of 3,464 from the territories concerned. Nearly half the students came from the West Indian territories. A total of 2,562 were at universities; 6,474 were training as nurses.

Hong Kong Medical Advisory Board Enlarged

The Hong Kong Medical Advisory Board, which was first set up in 1946 to advise the Governor on matters concerning the public health of the Colony, has been reconstituted with an enlarged membership and its terms of reference have been revised. The reconstituted board will now advise the Governor on matters of medical and health policy in Hong Kong as a whole. The Director of Medical and Health Services is *ex-officio* chairman, and the other four *ex-officio* members of the board are the vice-chairman of the Urban Council, the president of the Chinese Medical Association, the president of the Hong Kong Branch of the British Medical Association, and the principal medical and health officer, New Territories. The board also includes a medical officer of the armed Forces and a member of the Medical Faculty of the University of Hong Kong. Sir SIK-NIN CHAU, Dr. A. M. RODRIGUES, Dr. F. I. TSEUNG, and Dr. F. R. ASHTON are among those who have been appointed individually. The board's members will also include men closely connected with the medical work of traditional bodies and missionary groups as well as officials working in public medical and health services and those concerned in training doctors.

Medical Film Award

The Bronze Minerva Medal was recently awarded at the Fourth International Festival of Medico-Scientific Films at Turin to Mr. LAWRENCE ABEL and Mr. GORDON UNGLEY for the film "Haemorrhoids and the Early Detection of

Bronze Minerva Medal.

Cancer." The film, which is in 16 mm. colour and runs for nearly half an hour, was made under the auspices of the B.M.A., sponsored by W. R. Warner, Ltd., and produced and photographed by Stanley Schofield.

Poisons Information Centre, Leeds

A Poisons Information Centre has been set up in the receiving-room of the General Infirmary at Leeds. It houses an index file listing some 8,000 items that may be swallowed or inhaled, either by accident or design, at home or at work. These range from industrial substances, drugs, and plants to what is known in the trade as dry saltery goods: detergents, furniture polish, household disinfectants, and other cleaning agents. The index describes the toxic effects, if any, of the constituents of these products and the antidotes to be used in treatment. The file is available for reference on a 24-hour basis. The Infirmary's telephone number is Leeds 32799.

Presentation to Sir Austin Bradford Hill, F.R.S.

Old students of Sir Austin Bradford Hill gathered at the London School of Hygiene and Tropical Medicine on July 4 to attend a dinner in his honour and to make him a presentation before his forthcoming retirement from the chair of medical statistics in the University of London. Professor RICHARD SCHILLING spoke on behalf of the D.P.H. students and Mr. P. D. OLDHAM, statistician to the Pneumoconiosis Research Unit, on behalf of the "special course" students. Dr. RICHARD DOLL, who has collaborated with Sir Austin in many important investigations, acted as chairman and made the presentation of a cocktail cabinet, which was subscribed for by nearly 350 ex-students from all over the world. In his reply Sir AUSTIN BRADFORD HILL said he was deeply grateful to all those who had joined in this tribute to him, and wished that he could have thanked them all personally.

British Diabetic Association

Professor CHARLES BEST, F.R.S., was the guest of honour on July 6 at a luncheon to celebrate the fortieth anniversary of the discovery of insulin. The Minister of Health, Mr. J. ENOCH POWELL, in a speech of welcome said that the British Diabetic Association was the first of its kind in Britain. Dr. R. D. LAWRENCE, proposing Professor Best's health, said that $1m. had been raised in Canada this year for the Banting and Best Institute. The U.K. needed £100,000 for research in diabetes. In reply Dr. Best told many anecdotes that must have brought heart and encouragement to the many diabetics who attended the luncheon. The chair was taken by Sir BASIL HENRIQUES, who also presided on the same day at the 24th annual general meeting of the British Diabetic Association. Addressing the meeting, Professor Best emphasized the need for more money for research. "Diabetes," he said, "is downgraded in the minds of health authorities and the public because diabetics have kept their troubles too much to themselves." He urged more "shouting from the rooftops." He commended the British Diabetic Association for studying useful ways of uncovering unsuspected diabetes by "detection drives." As time went on we could expect more diabetics in view of the disease's inheritance. Finally, looking to the future, Professor Best forecast the day when not only should we have an oral insulin but even be able to interfere with the gene concerned in the carriage of diabetes.

"Abstracts of Efficiency Studies in the Hospital Service"

The Ministry of Health has issued to hospital authorities a second set of *Abstracts of Efficiency Studies in the Hospital Service*. The first set of seven studies was published in April. The second set of six studies covers maintenance of farms, grounds, and gardens; document copying; medical stenographers department; medical records (*a*) mobile steel shelving and (*b*) addressing machine system; and, finally, the collection of swill from hospital wards.

"Filing of Management Papers"

A report, *The Filing of Management Papers*, on the filing of papers relating to hospital administration prepared by the Hospital Organization and Methods Service of the Ministry of Health has been published by H.M.S.O., price 2s. 6d. The report describes weaknesses and suggests improvements in the handling and filing of such papers and brings together experience gained from studies undertaken for boards and committees at their request. It is the fifth in the series of organization and method reports that are being published. The earlier ones were *Out-patient Waiting Time* (No. 1); *Medical Records and Secretarial Services* (No. 2); *Chest Clinics* (No. 3); and *Organization and Management of Domestic Work in Hospitals* (No. 4).

London Medical Orchestra

At a recent concert given by the London Medical Orchestra a presentation was made to Dr. HANS UCKO, who started and ran the orchestra for seven years and who has now retired. He was recently made a Life Governor of the Imperial Cancer Research Fund in recognition of the large sum raised by the orchestra for cancer research. The orchestra meets every Thursday evening at the Rutherford School in Penfold Street, Edgware Road, and the conductor is now FREDERICK MARSHALL. Musical enthusiasts—dental or medical, pre- or postgraduate—are welcome as members, and should write to the secretary, Dr. H. O. ENGEL, 40 Warrington Crescent, London W.9.

Herta Oberheuser

HERTA OBERHEUSER, who was convicted at the Nuremburg war crimes trial of carrying out experiments on human beings at Ravensbruck concentration camp, has given up her claim to the title of doctor and the right to practise. (See *B.M.J.*, November 26, 1960, p. 1612, and December 10, 1960, p. 1748.)

In Brief

New out-patient and physiotherapy departments were officially opened at Hounslow Hospital, Staines Road, Hounslow, on July 11.

COMING EVENTS

British Medical Association: Sheffield Meeting.—Clinical Meetings will be held on July 20, 21, and 22. The full programme was printed in the *Supplement* of June 10.

Medical Art Society.—Annual exhibition of paintings, drawings, and sculpture at Leighton House Art Gallery, 12 Holland Park Road, London W.14, July 17 to August 5 (Monday to Friday, 11 a.m. to 7 p.m.; Saturday, 11 a.m. to 5 p.m.).

8th Annual Meeting, British Association of Paediatric Surgeons.—Stockholm, September 12–14, 1961. Secretary: Mr. R. B. ZACHARY, Institute of Child Health, Great Ormond Street, London W.C.1.

NEW ISSUES OF SPECIALIST JOURNALS

The journals listed below are obtainable from the Publishing Manager, B.M.A. House, Tavistock Square, London W.C.1.

THORAX

Traumatic Diaphragmatic Hernia. W. R. Probert and C. Havard.
A Review of Surgical Results in Hiatus Hernia. J. Leigh Collis.
A Study of the Chemical Composition and Potential Hazards of an Antifoam Substance Used in Intracardiac Surgery. J. S. Harington.
Empyema in Childhood. Eugene Hoffman.
Post-mortem Radiography and Gaseous Fixation of the Lung. R. J. R. Cureton and David H. Trapnell.
Diffuse Bronchopulmonary Aspergillosis. A. O. Bech.
Late Complications of Extraperiosteal Lucite Ball Plombage. M. Feuchtwanger, J. B. Borman, and I. Bruderman.
A Clinical Appraisal of Thymic Lesions and Anterior Mediastinal Lymphomata. B. T. Le Roux.
Fatal Spontaneous Rupture of an Aortic Aneurysm in a Young Adult. A. W. McCracken and D. J. Stoker.
Coarctation of the Aorta in Older Patients. F. T. I. Oey and J. A. Noordijk.
Pneumoconiosis Due to Fuller's Earth. A. Sakula.
Recurrence of "Benign" Intrathoracic Fibromas. W. F. Kerr and H. C. Nohl.
Stab Wounds Penetrating the Left Atrium. Milroy Paul.
Heterotopic Mediastinal Thyroid. B. T. le Roux.

Volume 16, No. 2. (Quarterly; £3 annually.)

BRITISH JOURNAL OF OPHTHALMOLOGY

The Diagnostic Value of Biomicroscopy of the Posterior Parts of the Eye. H. Goldmann.
The Eyes in Sarcoidosis. R. Pitts Crick, C. Hoyle, and H. Smellie.
Prevention of Experimental Ocular Hypertension with Polyphloretin Phosphate. D. F. Cole.
Ocular Changes Occurring during Cardiac Surgery under Profound Hypothermia and Occlusion. A. Lamb.
Mixed Glaucoma. J. D. Abrams.
Reviews.

Volume 45, No. 7. (Monthly; £6 annually. With *Ophthalmic Literature* and index, £9.)

JOURNAL OF NEUROLOGY, NEUROSURGERY AND PSYCHIATRY

Intrathecal Tuberculin in Disseminated Sclerosis: The Immunological Aspects. Honor V. Smith, Iwan E. Hughes, and George Hunter.
Therapeutic Trials in Multiple Sclerosis: Preliminary Report on Effects of Intrathecal Infection of Tuberculin (P.P.D.). Henry Miller, D. J. Newell, Allan Ridley, and Kurt Schapira.
Gamma Globulin Studies in Multiple Sclerosis and Their Application to the Problem of Diagnosis. Kurt Schapira and Dorothy C. Park.
The Stiffness of Spastic Muscle. John Foley.
Intense Rigidity of the Arms Due to Isolation of Motoneurones by a Spinal Tumour. Geoffrey Rushworth, W. A. Lishman, J. Trevor Hughes, and D. R. Oppenheimer.
The Effect of the Removal of the Nucleus Dentatus on the Parkinsonian Syndrome. Szabolcs Tóth.
Some Experimental Observations on the Action of Intravenous Hypertonic Urea in Dogs, with Particular Reference to Plasma Volume and Tissue Urea Changes. Shedden Alexander, J. C. Eaton, and H. J. Freedman.
On the Coexistence of Epileptic Seizures and Abnormal Involuntary Movements. Jerzy Chorobski and Tadeusz Bacia.
Narcolepsy in Identical Twins. Norman W. Imlah.
Observations on the Carotid Bruit. W. B. Matthews.
Herpes Zoster and the Landry-Guillain Barre Syndrome. J. D. E. Knox, R. Levy, and J. A. Simpson.
Hypersomnia in Dystrophia Myotonica. J. C. Phemister and J. M. Small.
The Fiction of the "Gerstmann Syndrome." Arthur L. Benton.
Vinyon "N" as Dural Substitute and Its Use in Other Neurosurgical Conditions. Paul Teng.
The Case Notes of the National Hospital for the Paralysed and Epileptic, Queen Square, London, Before 1900. Richard A. Hunter and I. J. Hurwitz.
Book Reviews.

Volume 24 (New Series), No. 2. (Quarterly; £3 annually.)

GUT

The Diagnosis and Treatment of Peptic Oesophagitis. Geoffrey Wooler.
The Turnover and Shedding of Epithelial Cells. B. Creamer, R. G. Shorter, and John Bamforth.
Part I. The Turnover in the Gastro-Intestinal Tract.
Part II. The Shedding in the Small Intestine.
The Influence of Cortisone on the Parietal Cell Population of the Stomach in the Dog. N. C. R. W. Reid, R. M. Hackett, and R. B. Welbourn.
The Measurement of Gastrointestinal Protein Loss by a New Method. K. N. Jeejeebhoy and N. F. Coghill.
The Stability of the Radioiriolein Bond in Intestinal Secretions. A. G. Cox and Z. Hinchliffe.
Proctalgia Fugax. Hassan Ibrahim.
Iron Deficiency after Partial Gastrectomy. J. R. Hobbs.
Some Metabolic and Haematological Effects of Oesophago-jejunostomy with By-pass of the Stomach. Sheila T. Callender, L. J. Witts, P. R. Allison, and A. Gunning.
A Clinical Appraisal of the Treatment of Chronic Duodenal Ulcer by Vagotomy and Gastric Drainage Operation. W. Gerald Austen and Harold C. Edwards.
Gastric Acid Secretion and Mucosal Appearances in Addison's Disease and Hypopituitarism. A. W. M. Smith, I. W. Delamore, and A. Wynn Williams.
Studies on the Site of Fat Absorption. 2. Fat Balances after Resection of Varying Amounts of the Small Intestine in Man. C. C. Booth, D. Alldis, and A. E. Read.
Methods and Techniques:
The Motility of the Pelvic Colon. 1. Motility in Normals and in Patients with Asymptomatic Duodenal Ulcer. A. M. Connell.
Potassium Replacement Therapy. G. N. Chandler, C. Hetherington, A. N. Stephenson, and M. Atkinson.
A Method of Measuring Gastric Pressure During Vagotomy. Peter A. Clarke.

Volume 2, No. 2. (Quarterly; £3 annually.)

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday, July 17

LONDON UNIVERSITY.—At Middlesex Hospital Medical School, 4 p.m., special university lecture in medicine by Professor R. L. Riley (Baltimore, U.S.A.): Interrelations Between Breathing and Blood Flow.

Wednesday, July 19

CAMBRIDGE UNIVERSITY: SCHOOL OF CLINICAL RESEARCH AND POSTGRADUATE MEDICAL TEACHING.—At Addenbrooke's Hospital (Postgraduate Lecture Room), 4.15 for 4.30 p.m., Sir Ernest Marsden, F.R.S.: Radioactivity of Soils, Food-plants, and Bones, With Special Reference to Niue Island.
ROYAL FREE HOSPITAL, Gray's Inn Road, W.C.—5.15 p.m., Dr. Robert M. Kark (Chicago): Pyelonephritis.
ROYAL SOCIETY OF HEALTH.—2.30 p.m., subject: Pollution of Underground Waters, Including the Recharging of Supplies. Papers by Dr. R. D. Maclean, Mr. J. C. Raffety, M.Inst.C.E., M.I.W.E., and Dr. S. Buchan, Ph.D.

Friday, July 21

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—3 p.m., Charles Tomes Lecture by Professor A. I. Darling: Selective Attack of Caries on the Dental Enamel.

APPOINTMENTS

BATES, J. A. V., M.B., B.Chir., Part-time Physician in Department of Applied Electrophysiology at the National Hospital, Queen Square, London W.C.

BIRMINGHAM REGIONAL HOSPITAL BOARD.—B. H. Bass, M.D., M.R.C.P., Part-time Consultant Physician to Lichfield, Sutton Coldfield, and Tamworth group of hospitals; Roger Morgan, M.B., B.S., D.P.M., Whole-time Director of Rehabilitation and Medical Superintendent to St. Wulstan's Hospital, Malvern.

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—P. G. I. Stovin, M.B., B.Chir., M.R.C.P., Consultant Pathologist, Papworth Hospital; S. K. Suppal, M.B., B.S., M.R.C.O.G., Registrar in Obstetrics and Gynaecology, West Norfolk and King's Lynn General Hospital; G. B. Markey, M.B., B.S., F.R.C.S.Ed., Surgical Registrar, Norfolk and Norwich Hospital; I. S. Trotter, M.R.C.P.Ed., Consultant Physician in Geriatrics, West Norfolk and North Cambridgeshire Area; K. Grainger, M.B., Ch.B., F.F.R., D.M.R.D., Consultant Radiologist, Ipswich and East Suffolk Hospital; P. K. M. Barrett, M.B., B.S., M.R.C.P., Senior Registrar in Chest Medicine, Cambridge Area Chest Clinic; E. P. Friday, M.B., Ch.B., Surgical Registrar, Newmarket General Hospital; J. B. Cowle, M.B., B.S., Ophthalmic Registrar, and I. H. Sayani, M.R.C.S., L.R.C.P., Orthopaedic Registrar, Norfolk and Norwich Hospital; Mrs. Sughra H. Asif, M.B., B.S., Registrar in Psychiatry, Hellesdon Hospital, Norwich.

GARDNER, P. S., M.D., Dip.Bact., Consultant Virologist to the United Newcastle upon Tyne Hospitals.

HOSPITAL FOR SICK CHILDREN, Great Ormond Street, London W.C.—Gunnar Schlistad, M.D., Sponsored House-surgeon; Veranna Kasetti, M.B., B.S., Resident Registrar to Ear, Nose, and Throat Department; E. F. Battersby, M.B., Ch.B., F.F.A. R.C.S., Anaesthetic Registrar; W. W. Slack, B.M., B.Ch., F.R.C.S., Surgical Out-patient Assistant; Muriel E. Rolfe, M.B., B.S., Part-time Medical Officer; L. J. Beilin, M.B., B.S., House-physician.

MANCHESTER REGIONAL HOSPITAL BOARD.—L. V. Gould, M.B., Ch.B., D.M.R.D., Consultant Radiologist (nine half-days weekly), Salford Group of Hospitals; H. L. Freeman, B.M., B.Ch., D.P.M., Consultant Psychiatrist (nine half-days weekly), Springfield Hospital and Salford Group of Hospitals; M Garretts, M.B., B.S., M.R.C.P., Consultant Dermatologist (five half-days weekly), South Manchester Group of Hospitals, Manchester and Salford Hospital for Skin Diseases, and Royal Manchester Children's Hospital (joint appointment with United Manchester Hospitals); D. M. Joshi, M.B., B.S., F.R.F.P.S., M.R.C.P.Ed., Whole-time Assistant Physician in Geriatrics, Bolton and District Group of Hospitals.

NORTH-WEST METROPOLITAN REGIONAL HOSPITAL BOARD.—J. D. B. Andrews, M.D., D.P.H., Consultant Physician, Geriatric Service, South-west Middlesex Group; J. M. Berg, M.B., B.Ch., Consultant Psychiatrist, Harperbury Hospital; A. G. Beckett, B.M., B.Ch., M.R.C.P., Consultant General Physician, Willesden General Hospital and Acton Hospital; Jean A Peachey, M.B., B.S., D.O., Ophthalmologist (S.H.M.O.), Barnet General Hospital and Middlesex County Council's Cranborne School Clinic, Potters Bar; Ann D. Black, M.B., Ch.B., D.P.M., Assistant Child Psychiatrist (S.H.M.O.), Hertfordshire Child Guidance Service; P. W. D. Corfield, M.S., F.R.C.S., Consultant General Surgeon, Harefield Hospital and Mount Vernon Hospital.

SOUTH-WESTERN REGIONAL HOSPITAL BOARD.—P. A. Skinner, M.B., B.S., F.F.A. R.C.S., Consultant Anaesthetist, Frenchay Hospital, Bristol; B. J. Muir, M.B., B.Chir., F.F.A. R.C.S., Consultant Anaesthetist, North Devon Infirmary, Barnstaple; F. J. Y. Wood, M.D., B.Chir., M.R.C.P., Consultant Physician with an interest in Geriatrics, South Somerset Clinical Area; E. M. Edwards, M.B., M.S., M.R.C.O.G., Consultant Obstetrician and Gynaecologist, North Gloucestershire Clinical Area; M. A. Voyce, M.B., Ch.B., Medical Registrar, Bath Group of Hospitals; G. Corney, M.B., Ch.B., D.Obst.R.C.O.G., D.C.H., Medical Registrar, Bath Group of Hospitals; S. K. Basu, M.B., D.A., Anaesthetic Registrar, Southmead Hospital, Bristol.

THOMAS, D. VERNON M.D., F.F.A. R.C.S., Associate Professor of Anaesthesia, Stanford University School of Medicine, U.S.A., as from September 1, 1961.

THOMAS, L. P., M.Ch., F.R.C.S., Consultant Surgeon, Royal Gwent Hospital, Newport.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Staniland.—On July 4, 1961, at Christchurch, New Zealand, to Shelagh Staniland (formerly Mathias), M.B., B.S., D.A., wife of Gerald Cedric Staniland, M.Sc., F.R.C.S.—a son.

DEATHS

Bayly.—On June 22, 1961, at his home, Camberley, Surrey, Maurice Beddow Bayly, M.R.C.S., L.R.C.P., aged 74.
Bryden.—On June 19, 1961, at Salisbury, Ronald Anderson Bryden, D.S.O., M.R.C.S., L.R.C.P., Colonel, R.A.M.C., retired, aged 78.
Buckley.—On June 17, 1961, at St. Bartholomew's Hospital, Charles Dudley Maybury Buckley, *M.C.*, M.B., B.Ch., Colonel, late R.A.M.C., retired, of Heather House, Fleet, Hants.
Campbell.—On June 17, 1961, at Erskine Hospital, Bishopton, Charles Campbell, L.R.C.P.&S., D.P.H.
Corkery.—On June 4, 1961, Martin Percy Corkery, O.B.E., L.R.C.P.&S., L.R.F.P.S., Lieutenant-Colonel R.A.M.C., retired.
Fuller.—On June 17, 1961, Frederick Holcombe Fuller, M.R.C.S., L.R.C.P., L.D.S., of Lewes, Sussex, late of 44 Brunswick Square, Hove, Sussex, aged 78.
Johnson.—On June 15, 1961, the result of an accident, William Lawrence Johnson, M.B., B.Ch., late of Chesham, Bucks.
Macinnes.—On June 21, 1961, at Tyldesley, Lancs, Alexander Macinnes, M.B., Ch.B.
Parfitt.—On June 12, 1961, at Greenhill, Chidcock, Bridport, Dorset, John Broadribb Parfitt, M.R.C.S., L.R.C.P., L.D.S. R.C.S., aged 89.
Probyn-Williams.—On June 13, 1961, at Chipping Manor, Wotton-under-Edge, Glos, Robert Cuthbert Probyn-Williams, B.M., B.Ch.
Singer.—On June 22, 1961, Kildare Lawrence Singer, M.R.C.S., L.R.C.P., of Botwell Common Road, Hayes, Middlesex.
Smyth.—On June 18, 1961, David William Smyth, M.B., B.Ch., D.Obst. R.C.O.G., of Moorfield House, Hereford.
Woods.—On June 19, 1961, Matthew May Woods, M.R.C.S., L.R.C.P., of The Rowans, Redcot Lane, Surry, Kent.
Wotherspoon.—On July 16, 1961, at Beckenham Hospital, John Wotherspoon, M.D., D.P.H., D.T.M.&H., of 2 The Knoll, Beckenham, Kent, aged 85.

Any Questions ?

We publish below a selection of those questions and answers which seem of general interest. It is regretted that it is not possible to supply answers to all questions submitted.

Excessive Glutinous Saliva

Q.—*Is there any known factor which would cause the mucinous rather than the serous salivary glands to be stimulated, and so cause a patient to have excessive glutinous saliva? Is there any treatment for this condition?*

A.—Saliva consists of two components—a serous or watery secretion and a mucinous or sticky secretion. Of the three main salivary glands the parotid secretes only watery saliva, the submaxillary gland may secrete either watery or mucinous saliva, and the sublingual gland produces mainly mucinous saliva. The control of salivation is through the autonomic nervous system, and in general terms it can be said that in animal experiments stimulation of the sympathetic causes the secretion of a small amount of viscous saliva and stimulation of the parasympathetic (chorda tympani nerve) produces a copious secretion of watery saliva. The normal stimuli for salivation are the gustatory, olfactory, and psychic influences associated with the taking of food. Both the amount and composition of the saliva depend upon the nature of the stimulus. Most foods which excite the appetite produces a moderate flow of viscid saliva, whereas dry or irritant substances in the mouth cause a copious flow of watery saliva.

If the patient is suffering from excessive salivation this is most likely reflex in origin and due to some local lesion in the upper gastrointestinal tract. The fact that the secretion is thick and glutinous indicates that it is coming mainly from the submaxillary and sublingual glands, and suggests the possibility that the patient may have some local lesion in the mouth which is causing reflex sialorrhoea and is also causing occlusion of the parotid ducts. As indicated in a previous answer in this column,[1] the first approach to treatment should be to look for some local lesion in the upper gastrointestinal tract and to deal with it appropriately. If this is unsuccessful then atropine or one of the other belladonna alkaloids or their analogues should be tried, as these are the best drugs for inhibiting the secretion of saliva. It is to be noted, however, that the amount of these drugs required to cause dryness of the mouth may be much larger than the usual pharmacopoeial dose. It might also be worth while trying one of the ganglion-blocking agents such as pempidine or mecamylamine; experience with these drugs in the treatment of hypertension indicates that even in small doses they cause considerable dryness of the mouth.

REFERENCE

[1] *Brit. med. J.*, 1960, 2, 83.

Nystagmus

Q.—*Is there any accepted definition which distinguishes fine from coarse nystagmus? How frequently does nystagmus or nystagmoid movement occur in normal people?*

A.—Fine and coarse nystagmus are descriptive terms which lack clear definition or any standard of measurement. Normal people may manifest nystagmus under certain conditions—when they watch a succession of rapidly moving objects (opticokinetic nystagmus, seen in the subject gazing from the window of a moving train), on forced rotation in a revolving chair, on douching an external auditory meatus with hot or cold water, or under the influence of such drugs as alcohol, barbiturate, or quinine. Nystagmus is also seen without a gross structural cause in the blind, in albinism, in those who have long worked in a dim light, and in several members of some families. However, what the questioner probably has in mind is the fact that on routine neurological examination of otherwise healthy patients a few irregular jerks of the eyes often occur on extreme lateral fixation: these are often referred to as "nystagmoid jerks." Such a phenomenon is certainly within the range of normality. It is distinguished from nystagmus of clinical significance by the fact that it is neither regular nor sustained.

Stop: You Have Been Warned

Q.—*What is the maximum dose of (1) gin, (2) sherry, and (3) champagne which can be taken on an empty stomach without raising the blood alcohol concentration above 50 mg. per 100 ml.?*

A.—The quantity of any alcoholic beverage required to produce a given blood alcohol concentration varies widely. It depends mainly on the weight of the person taking it and on the degree of absorption from the alimentary canal. Even under standard conditions there is variation from individual to individual, but average figures for an 11-stone (69.9-kg.) man taking alcohol on an empty stomach can be given. To provide a concentration of 50 mg. per 100 ml. this would require about 36 ml. of absolute alcohol, which is contained in 90 ml. (3¼ oz.) of gin, 185 ml. (6½ oz., or about a quarter of a bottle or four glasses) of sherry, or 350 ml. (13 oz.) of champagne. The minimum possible amounts which in a person absorbing the alcohol very rapidly and completely could give rise to a concentration of 50 mg. per 100 ml. of blood are much less. These would be, in an 11-stone man, 45 ml. (1¾ oz.) of gin, 95 ml. (3¼ oz. or two glasses) of sherry, and 180 ml. (6½ oz.) of champagne. Maximum figures cannot be given: when alcohol is taken on an empty stomach it would be unsafe to assume that they are much higher than the average figures quoted above.

First-aid Book for Laymen

Q.—*An intelligent layman with no medical knowledge asks me to recommend a fairly comprehensive book on first aid for him to take abroad to an area where medical assistance will be difficult. Could you advise me on such a book?*

A.—One of the best and certainly the best known is the manual published by the St. John Ambulance Society, the St. Andrew's Ambulance Society, and the British Red Cross Society. It has been brought up to date and costs 4s. A slightly more comprehensive book, still of small size, is Baillière's *Handbook of First Aid* by Belilios, Mulvany, and Armstrong. The price is 8s. 6d. *First Aid in the Factory*, written by Lord Taylor, is of more general application than its title suggests, and is very attractively produced, price 9s. 6d. A much larger book, not meant for laymen, but containing a great deal of "first-aid" information, is the *Handbook for the Royal Naval Sick Berth Staff*, BR 888, published by H.M. Stationery Office at the price of 30s.

Diphtheria Immunization in Older Children

Q.—*Which is the best preparation, and in what dosage, to use in a child aged 13 who has not been immunized against diphtheria since the age of 5?*

A.—With children over the age of 8–10 years, it is desirable to carry out a preliminary Schick test: this obviates the untoward consequences of injecting diphtheria vaccine into Schick "pseudo" reactors. The early reading of Schick tests (at 48 hours) should not be omitted, because of the danger of missing "pseudo" reactors at the 5–7 day reading. These subjects are invariably immune, even when the reaction on the test arm is somewhat greater than that on the control. The choice of preparation for the non-immune older child is between diphtheria formol toxoid (Dip/Vac/FT) and diphtheria toxoid-antitoxin floccules (Dip/Vac/TAF), and is largely a personal one. F.T. is thought by some practitioners to give rather more reactions,

while T.A.F. is avoided by others because it contains a small amount of horse serum to which a person might be sensitized. A full reinforcing dose of either preparation should give adequate protection provided that the primary course at the age of 5 was complete. The modern trend, however, is towards the use of combined vaccine whenever possible. If the child has already been actively immunized against tetanus, diphtheria and tetanus vaccine (DT/Vac) is an excellent prophylactic for reinforcement.

Spot Weight Reducing

Q.—*Is there any scientific basis for the claims that so-called spot-reducing garments (made of plastic materials) "dissolve fatty tissue" in selected areas of the body by causing excessive local perspiration? Is local vibratory treatment of any value in reducing fat in selected parts of the body?*

A.—Apparent fatness may be due to two causes: (1) gain in weight; (2) lack of tone of the abdominal musculature. There is no scientific basis for claims that excess weight will be removed by any form of physiotherapy or any type of garment. A lax abdominal wall will be concealed by a spot-reducing garment or by any other form of corset. It can be remedied by suitable exercise therapy. Vibratory treatment may help indirectly by making the patient conscious of his abdominal muscles and therefore reminding him to contract them.

Nasal Furuncles

Q.—*What can be done for recurrent furuncles in the nose? The patient is allergic to penicillin, and the regular application of "naseptin" cream has been ineffectual.*

A.—Perhaps the most common cause of recurrent furuncles in the nose is physical irritation to the nostrils, especially by the fingers. Diabetes should always be excluded, as also should chronic sinus infection and nasal allergy. Trimming of the nasal vibrissae may help, as also may the local application of hydrocortisone cream or dilute nitrated mercury ointment. It is, of course, of the utmost importance that no attempt should be made to pull out the hairs, since the nostrils come within the "danger area" of the face.

Excessive Milk Consumption and Respiratory Infections

Q.—*Is there any proved relationship between excessive (1½–3 pints [0.9–1.7 litres] a day) consumption of cow's milk in young children and (1) frequency of upper respiratory infections, and (2) hypertrophy of tonsils and adenoids?*

A.—There is no known relationship between the excessive consumption of cow's milk and the frequency of upper respiratory infections or hypertrophy of tonsils and adenoids.

Post-herpetic Facial Neuralgia

Q.—*A man aged 55 has severe post-herpetic neuralgia involving the right 5th cranial nerve and complete sensory loss on the right side of the face. The ganglion on the right side has been injected. Is there any likelihood that neurosurgery would help this patient, and, if so, what would be the procedure?*

A.—The complete sensory loss on the right side of the face presumably results from the Gasserian ganglion injection rather than from the attack of herpes zoster. Ganglion injection is only very rarely successful in relieving post-herpetic neuralgia, unlike the excellent results obtained in paroxysmal trigeminal neuralgia. The same is also true of operative section of the sensory root of the trigeminal nerve and of trigeminal tractotomy in the medulla, and these procedures would not be indicated in the case mentioned. Long-standing post-herpetic neuralgia is a notoriously difficult condition to treat, but Taverner[1] has recently claimed encouraging results with brief interrupted spraying of the affected area with ethyl chloride. If, however, his explanation that the relief of pain by cooling the skin depends on adequate afferent stimulation is correct, it is unlikely to be effective in a patient in whom the skin has already been denervated.

REFERENCE

[1] Taverner, D., *Lancet*, 1960, **2**, 671.

NOTES AND COMMENTS

Withdrawing C.S.F. from Spitz–Holter Valves.—Dr. R. P. CALLAGHAN (London N.W.6) writes: Your expert ("Any Questions?" June 3, p. 1622) has pointed out why a Spitz–Holter valve should not be punctured to obtain a specimen of C.S.F. However, the indications to investigate this fluid warrant particular attention, because, unlike other drainage operations of hydrocephalus—viz., spinoureteric,[1] ventriculo-subdural,[2] etc.—the shunting of C.S.F. through valve systems either into the right atrium[3] or into the superior vena cava[4] provides an unguarded access for organisms to enter the circulation. It is therefore appropriate to suggest that persistent bacteriaemia[5] may be the cause of this child's symptoms which the questioner is investigating. In this condition organisms of low virulence, frequently the coagulase-negative *Staphylococcus albus*, colonize the valve. By virtue of their low virulence, they do not cause obvious clinical inflammation around the valve but are carried by the flow of C.S.F. through a functioning valve system into the heart to produce persistent bacteriaemia, characterized by persistent pyrexia, progressive anaemia, and splenomegaly. Blood cultures are positive, but, since the offending organism is frequently a *Staph. albus*, there may be a tendency to regard this essential finding as an indication of a contaminated blood culture. Antibiotics given intraventricularly and systemically can only suppress the symptoms.[6] Mr. G. H. Macnab, at the Hospital for Sick Children, Great Ormond Street, has had seven of these cases in a series of over 120 Spitz–Holter valves, and it has been found that the condition cannot be cured until the valve is removed or disconnected.

OUR EXPERT replies: Dr. Callaghan's comment goes beyond the terms of the original question, but he is quite correct in saying that the Spitz–Holter valve by itself can lead to a meningitis from organisms of low virulence and can perpetuate this meningitis. Indeed, this seems to be a quite serious complication of the valve, and if the meningitis does occur the treatment of the meningitis is not aspiration of C.S.F. by any route but by removal of the valve.

REFERENCES

[1] Matson, D. D., *J. Neurosurg.*, 1949, **6**, 238.
[2] Forrest, D. M., Laurence, K. M., and Macnab, G. H., *Lancet*, 1957, **1**, 1274.
[3] Pudenz, R. H., Russell, F. E., Hurd, A. H., and Shelden, C. H., *J. Neurosurg.*, 1957, **14**, 171.
[4] Spitz, E. B., *Symposium on Pediatric Surgery*, 1957, **6**, 1215.
[5] Cohen, S. J., and Callaghan, R. P., *Brit. med. J.*, 1961, in press.
[6] Callaghan, R. P., Cohen, S. J., and Stewart, G. T., ibid., 1961, **1**, 860.

Correction.—We much regret an error in Mr. Lindsay Symon's preliminary communication (July 8, p. 94). In lines 10 and 11 the doses of isotope recommended by Selverstone and White (1951) and Selverstone and Moulton (1957) should have read, respectively, "0.5 to 1 mc." and "1 mc. or less."

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